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ROY WALDO MINER

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*SECTION OF PHYSICS AND CHEMISTRY**

THE CORROSIVITY OF FUMING NITRIC ACID**

By John D. Clark and Michael E. Walsh

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Introduction

Fuming nitric acid is an oxidizer frequently used for rocket fuels because of its high percentage of available oxygen, its high density, which allows a large quantity of oxidant to be carried in a relatively small tank, and because of its low cost and ready availability. It is used either as white fuming nitric acid (WFNA), which contains no more than 2 per cent H_2O and perhaps as much as 2 per cent NO_2, or as red fuming nitric acid (RFNA), which generally contains 2 or 3 per cent water and and may contain as much as 20 to 25 per cent NO_2.

Neither red nor white fuming nitric acid has much in common with the so-called "concentrated", or 70 per cent acid used in most laboratories. The corrosion problems, in particular, are of an entirely different order of magnitude. Seventy per cent acid can be contained indifinitely in the 300 series stainless steels, but WFNA or RFNA, after a week or two in an SS-347 drum at room temperature, is literally black with corrosion products.

As a part of a recently completed project, the Chemistry Branch of the United States Naval Air Rocket Test Station (NARTS) was assigned the task of investigating the nature and rate of this corrosion, with the eventual object of reducing corrosion to a tolerable amount.

Procedure

Two series of corrosion measurements were made. The first was upon a nitric acid containing 23 per cent NO_2 and 2 per cent H_2O, at three different temperatures, 3° C, 30° C, and 60° C. The second was carried out at 3° C, upon a series of acids of various compositions containing, initially, 0, 2.5, or 5 per cent H_2O and 0 or 10 per cent NO_2. The metal investigated was in all cases SS-347, an 18-8 Austenite stainless steel stabilized with niobium. The red acid runs were made in Teflon-lined stainless steel bombs, pressurized with 25 p.s.i.g. of O_2 in order to inhibit the autodecomposition of the acid. Both liquid– and gas–phase corrosion were measured. The runs on varying acids were made in glass at atmospheric pressure. Only liquid-phase corrosion was measured. In all cases, the liquid volume/sample area ratio was held at 50 cc./in.^{2},

*The Section of Geology and Mineralogy held a meeting on January 3, 1955 at which a paper entitled "Sedimentation in the Persian Gulf" was read by Brooks F. Ellis. No abstract of this paper has been received.

**This paper, illustrated with lantern slides, was presented at a meeting of the Section on January 4, 1955.

approximately the ratio obtaining in a 50-gallon drum.

Triplicate samples (strips of metal 1 × 1-3/4 × 1/16 inches) were held in, or suspended above, the acid in every case. The corrosion was determined by periodically removing, washing, drying, and weighing the samples. The checks among the triplicate samples, incidentally, were excellent. The runs were from 35 to 260 days, depending upon the corrosion rate.

Experimental Data

Red acid runs. The liquid-phase corrosion of SS-347 in the 23 per cent NO_2 acid is shown in FIGURE 1, in which the corrosion is shown as milligrams weight loss per square inch of surface. Two things are immediately obvious: the very high temperature coefficient of corrosion and the sudden cessation of corrosion after 15 days in the 60° C. run. The latter phenomenon may be explained quite simply by the build-up of corrosion products in the acid. The acid, including the corrosion products from the gas-phase samples (which were suspended above the acid in which the liquid-phase samples were immersed), was approximately 0.15 normal in metal nitrates by the 15th day, and it would not be surprising to find the concentration of the corroding species, whatever its nature, reduced by the presence of that much nitrate.

FIGURE 2 shows the gas-phase corrosion of SS-347 above the 23 per cent NO_2 acid. Here, the temperature coefficient is even higher, and the corrosion does not drop off, since, obviously, metallic nitrates cannot accumulate in the vapor phase. There was no formation of a protective coating in any case, and no appreciable sludge formation. A powdery black coating on the specimens, easily washed off, was shown by a qualitative test to be largely MnO_2.

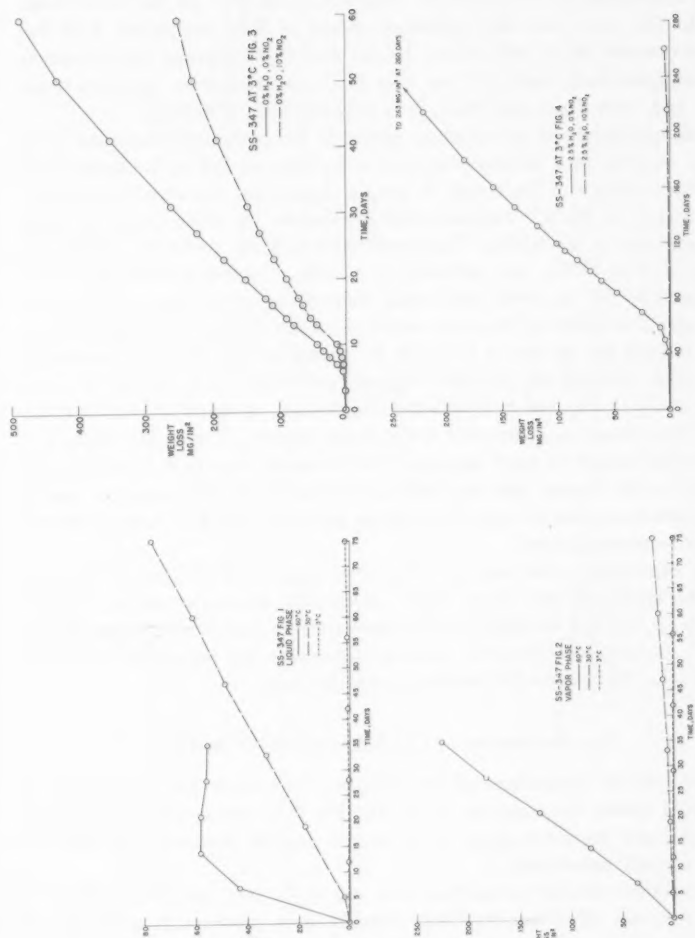
3°C. Runs with different acids. FIGURE 3 shows the effect of NO_2 upon the corrosivity of completely anhydrous HNO_3. The low temperature of the runs (3°C.) slowed down the corrosion until the process could be followed in some detail. The eventual decline in corrosion rate due to accumulation of corrosion products is clearly visible, as is an apparent induction period before the corrosion reaches its full rate.

FIGURE 4 shows the corrosivity of HNO_3 containing 2.5 per cent H_2O, with and without the addition of 10 per cent NO_2. In this case, not only is the corrosion rate much lower than in the dry acid, but the inhibiting effect of NO_2 is much greater. The induction period is again apparent.

FIGURE 5, in turn, shows the effect of NO_2 upon the corrosivity of acid containing 5 per cent H_2O. The same trends are apparent here.

The most striking features of these data are:

(1). The general similarity of the corrosion-time curves whenever the corrosion has progressed to any considerable degree—an S curve with a short lower limb.


SS-347 AT 3°C FIG. 3
0.5% H2O2, 0.5% HCl
0.5% H2O2, 0.05% HCl
WEIGHT LOSS MG/in²
TIME, DAYS
SS-347 AT 3°C FIG. 4
2.5% H2O2, 0.5% HCl
2.5% H2O2, 0.05% HCl
WEIGHT LOSS MG/in²
TIME, DAYS
SS-347 FIG. 1
LIQUID PHASE
60°C
30°C
3°C
WEIGHT LOSS MG/in²
TIME, DAYS
SS-347 FIG. 2
VAPOR PHASE
60°C
30°C
3°C
WEIGHT LOSS MG/in²
TIME, DAYS

(2). The magnitude of the inhibitory effect of H_2O upon corrosion. In 59 days, acid containing no water corroded about 485 mg./in.^{2}, while acid containing 2.5 per cent H_2O corroded only 263 mg./in.^{2} in 260 days. Acid containing 5 per cent H_2O corroded 16 mg./in.^{2} in the same time.

(3). The fact that the inhibitory effect of NO_2 increases with the water content. With zero water, 10 per cent NO_2 reduced the corrosion by not quite half; with 2.5 per cent H_2O, the reduction is close to 60-fold; and, with 5 per cent H_2O, it is approximately 200-fold.

The appearance of an induction period in the corrosion raised the point as to whether this phenomenon was a function of the acid composition or of the surface of the metal. A simple experiment answered this question. Strips of SS-347 were allowed to corrode for three days, at room temperature, in dry HNO_3. They were then washed, dried, and placed in *fresh* acid at 30°C., and allowed to corrode. Control specimens of undamaged SS-347 in fresh acid were treated the same way, as were undamaged specimens in the acid which had been used in the pretreatment. The results are shown in FIGURE 6. From the fact that the pretreated specimen showed by far the highest corrosion rate, while the new specimens in the old acid showed the lowest, it seems clear that the induction period is a function of the metal surface. Either the surface of the rolled strips is more resistant to corrosion than is the bulk of the metal, or the lesser area available for corrosion on the initially smooth strip forces a reduced rate of corrosion until the area has been increased by the corrosion itself.

An experiment was made in which two sets of SS-347 strips (one set sandblasted and the other highly polished), were allowed to corrode (FIGURE 7). The corrosion curves demonstrate that a roughening of the metal, increasing its initial surface, decreases the induction period but has little effect upon the eventual corrosion rate.

The Mechanism of Corrosion of SS-347 in HNO_3

The critical dependence of the corrosion rate upon the composition of the acid raises the question as to whether the identity of the corroding species and the mechanism of its action can be deduced from the raw data already presented.

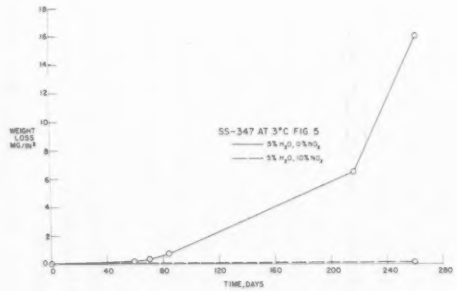
The three possible oxidizing ions are $H_3\overset{+}{O}$, $N\overset{+}{O}$, and $N\overset{+}{O}_2$. The fact that the rate of corrosion drops sharply with increasing water would appear to rule out the hydronium ion, since, by the equilibrium

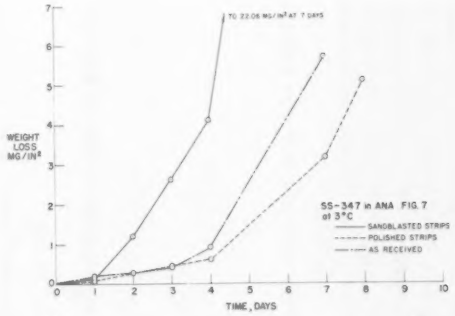
$$HNO_3 + H_2O = H_3\overset{+}{O} + N\bar{O}_3$$

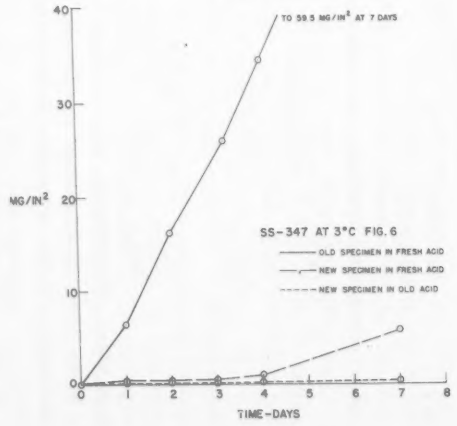
the increasing water should increase the $H_3\overset{+}{O}$ concentration. Nor does $N\overset{+}{O}$ appear to be the major agent, since it is in equilibrium with NO_2

$$2NO_2 = N_2O_4 = N\overset{+}{O} + N\bar{O}_3,$$

and increasing the NO_2 should increase the $N\overset{+}{O}$ concentration and, contrary to the experimental facts, should increase the corrosion rate.


SS-347 AT 3°C FIG. 5
5% H2O, 0% NO3
5% H2O, 10% NO3
WEIGHT LOSS MG/IN2
TIME, DAYS


TO 22.06 MG/IN2 AT 7 DAYS
SS-347 in ANA FIG. 7 at 3°C
SANDBLASTED STRIPS
POLISHED STRIPS
AS RECEIVED
WEIGHT LOSS MG/IN2
TIME, DAYS


TO 59.5 MG/IN2 AT 7 DAYS
SS-347 AT 3°C FIG. 6
OLD SPECIMEN IN FRESH ACID
NEW SPECIMEN IN FRESH ACID
NEW SPECIMEN IN OLD ACID
MG/IN2
TIME-DAYS

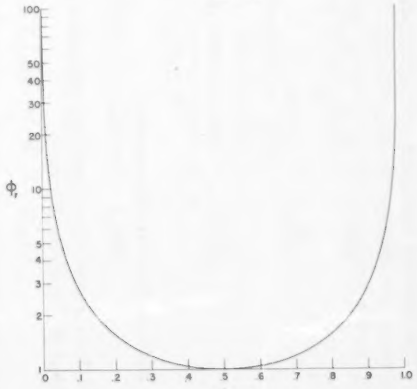

Φ7
Y/(X+Y)

FIG. 8

It appears probable that the nitryl or nitronium ion, NO_2^+, is the active corroding agent. In order to confirm this hypothesis quantitatively, it is necessary to get some idea of the nitronium content in acids containing different amounts of H_2O.

The relevant equilibria are as follows:

$$2HNO_3 = N^+O_2 + H_2O + N\bar{O}_3$$

$$HNO_3 + H_2O = H_3^+O + N\bar{O}_3$$

or

$$(N^+O_2) = \frac{A(HNO_3)^2}{(N\bar{O}_3)(H_2O)}$$

$$(H_3^+O) = \frac{B(H_2O)(HNO_3)}{(N\bar{O}_3)}$$

In addition there is the equation:

$$(NO_2^+) + (H_3^+O) = (N\bar{O}_3)$$

which expresses the electrical neutrality of the solution and, if W is the added or "analytical" water,

$$(H_2O) = W + (NO_2^+) - (H_3^+O),$$

since the formation of one NO_2^+ produces a molecule of water and that of one H_3^+O consumes one.

Now, assuming that (HNO_3), the major species, remains relatively constant, we can make the substitutions:

and

$$A\ (HNO_3)^2 = a$$

$$B\ (HNO_3) = b$$

Four equations involving four unknowns can now be set up:

$$(NO_2^+) = \frac{a}{(N\bar{O}_3)(H_2O)}$$

$$(H_2O) = \frac{b(H_2O)}{(N\bar{O}_3)}$$

$$(H_2O) = W + (NO_2^+) - (H_3^+O)$$

$$(N\bar{O}_3) = (NO_2^+) + (H_3^+O)$$

from which, theoretically, the nitronium concentration can be derived.

When, however, we attempt to solve for this value ($x = (NO_2^+)$) we are confronted with the malevolent-looking expression:

$$x\left(x-b+\sqrt{(b+x)^2+4b(w+x)}\right)\left(3x+2w+b+\sqrt{(b+x)^2+4b(w+x)}\right) = 2a$$

containing two unknown constants, *a* and *b*, and are immediately convinced that such an approach is unlikely to be profitable.

It is possible, however, to relate the ionic content of the acid to its electrical conductivity by the equation

$$F = C_1(\bar{NO_3}) + C_2(\overset{+}{H_3}O) + C_3(\overset{+}{NO_2}).$$

Since $$(\bar{NO_3}) = (\overset{+}{H_3}O) + (\overset{+}{NO_2})$$

and since the hydronium ion and the nitronium ion are not too different in size and should have similar mobilities, and since the nitrate ion, like the hydroxyl ion in aqueous systems, can be confidently expected to have an apparent mobility much higher than either of the others, the conductivity can be approximated by the equation

$$F = h(\bar{NO_3}).$$

Starting now with the same expressions as before

$$(\overset{+}{NO_2}) = \frac{a}{(\bar{NO_3})(H_2O)} \quad \text{and} \quad (\overset{+}{H_3}O) = \frac{b(H_2O)}{(\bar{NO_3})}$$

plus $$(\bar{NO_3}) = (\overset{+}{NO_2}) + (\overset{+}{H_3}O), \text{ and } F = h(\bar{NO_3})$$

and letting $(\overset{+}{NO_2}) = x$ and $(\overset{+}{H_3}O) = y$, as before, we get eventually

$$\frac{(x+y)^2}{xy} = \frac{F^4}{abh}.$$

The expression $\frac{(x+y)^2}{xy}$, designated by ϕ, is dimensionless, is a function of, and *only* of, the ratio between x and y, minimizes at $x = y$, and approaches infinity when x or y approaches zero. Finally, it is proportional to the fourth power of a measurable parameter, the conductivity.

The conductivity of HNO_3, containing various percentages of water, has been measured at 0° C. by J.P.L.[1] and, at the high HNO_3 end, it minimizes at approximately 3.2 per cent H_2O. At this point, therefore, the concentration of the hydronium ion equals that of the nitronium ion. It is now convenient to define a new function, $F_r = F/F_{min.}$ If $\phi_r = \phi/\phi_{min.}$ is now plotted against $y/x+y$ (FIGURE 8), a determination of the ratio of the hydronium ion concentration to the total cation concentration at any point on the conductivity curve is possible. For instance, at 1.2 per cent H_2O the conductivity is 199 $(\text{ohm-cm.})^{-1}$. The minimum conductivity is 131 $(\text{ohm-cm.})^{-1}$, so $F = 1.5191$ and $F^4 = 5.225$. Consulting the ϕ_r curve, we find that $y/x+y = 0.050$. The conductivity of HNO_3 at 0 per cent H_2O is 377 $(\text{ohm-cm.})^{-1}$. Taking this as equivalent to a unit total concentration of cations, the cationic concentration, the $(\overset{+}{H_3}O)$ concentration (total cations x $y/x+y$), and $(\overset{+}{NO_2})$ concentration (total cations minus $\overset{+}{H_3}O$) can all be plotted in terms of this unit as a function of water content (FIGURE 9). The relative $(\overset{+}{NO_2})$ concentration can now be found for any acid of interest.

It is now possible to investigate the dependence of the corrosion rate

upon (NO_2^+). Let us consider the three NO_2-free acids, with 0, 2.5, and 5 per cent H_2O. From the corrosion curves we can determine the maximum rate of corrosion after the induction period and before the accumulation of products has slowed down the process.

If we plot the log of the relative rate against two thirds of the log of the relative (NO_2^+) concentration (to change volume to surface concentration), we get the results shown in FIGURE 10. The best line through these points has a slope of 1.95, which is so close to two as to make inevitable the conclusion that we have to do with a second order reaction.

It is suggested then, that the controlling reaction in the corrosion process is

$$Fe + 2\,NO_2^+ \rightarrow Fe^{++} + N_2O_4.$$

The presence of ferrous iron in fuming nitric acid may be surprising, but Fe^{++} in high concentrations has frequently been observed in iron-contaminated acids in the NARTS laboratory. Likewise, in the RFNA bomb runs, whenever an SS-347 sample had been seriously corroded, it was noticed that the O_2 pressure originally placed upon the acid had vanished when the bomb was opened, as might be expected if any considerable quantity of ferrous iron were available to react with the oxygen.

A start may be made, now, in setting up an equation for the corrosion rate of SS-347 in HNO_3.

$$R = (NO_2^+)^{(2 \times 2/3)}\ a\, e^{\frac{-\Delta h}{RT}}$$

containing the two constants a and Δh. Let us consider the latter—the heat of activation of the process.

The bomb runs were made with RFNA, but if the nitronium ion is the corroding species, Δh should be the same with RFNA as with WFNA. Making this assumption, an appreciable amount of data are available. The liquid phase rates at 60°C. and 30°C. (the 3°C. rate is so low that the data are unreliable) and the vapor phase rates at 60°, 30°, and 3°C. In the liquid phase, the concentration of the nitric acid is not a function of temperature, and that of the corroding species (NO_2^+), presumably, also remains constant, so that the liquid phase data may be used directly. In the gas phase, the situation is otherwise, since the surface concentration of the nitronium ion should vary as the two thirds power of the nitric acid vapor pressure, and the corrosion varies as the second power of this surface concentration. Hence, the rates observed must be divided by the four-thirds power of the vapor pressure of the nitric acid to be comparable with the liquid-phase data. When this is done, the results are shown in FIGURE 11, where relative rates are used as before. The near

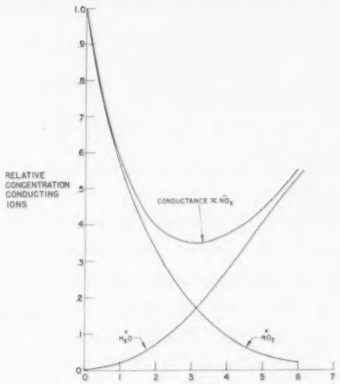


FIG. 9

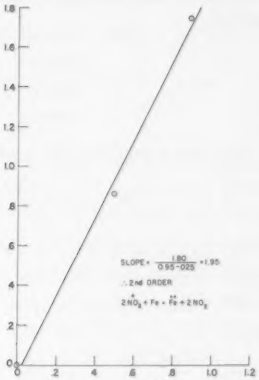


FIG. 10

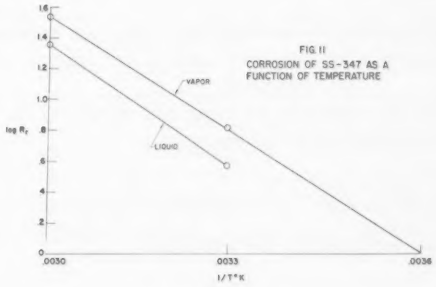


FIG. 11
CORROSION OF SS-347 AS A FUNCTION OF TEMPERATURE

parallelism between the liquid and the vapor lines is remarkable and encouraging.

Δh may now be derived by the standard method, with the following results.

Δh	liquid	30°C.-60°C.	12.05 Kcal./mol
	vapor	3°C.-30°C.	11.53 Kcal./mol
	vapor	30°C.-60°C.	11.11 Kcal./mol
	average		11.6 ± 0.5 Kcal./mol

The rate equation may now be written

$$R = (\overset{+}{NO_2})^{\frac{4}{3}} \quad a\, e^{\frac{-11,600}{RT}}$$

which leaves only *a* to be evaluated.

This parameter may be estimated as follows: the corrosion rate of SS-347, after the induction period, at 3°C., in pure HNO_3, is approximately 12 mg./in.^{2} per day or 34 mils per year. Taking Ingold's[2] value for nitronium ion in pure HNO_3 (1.67 mol per cent, or 0.404 normal) and substituting, we can solve for *a*, getting the final explicit equation, where R is in mils per year, and concentration is in volume normal:

$$R = 1.57 \times 10'' \ (NO_2^+) \ e^{\frac{-11,600}{1.987T}}$$

This equation, at present, can be used quantitatively only with white acids, since only in the case of acids containing a negligible amount of NO_2 can we solve for the nitronium content. It does, however, explain many cases of corrosion reduction since if $\overset{+}{NO_2}$ is the corroding ion, there will be a corresponding reduction in corrosion whenever its concentration is reduced by the addition of water, of NO_2, of soluble nitrates, or otherwise.

Thus, a large quantity of heterogeneous corrosion data can be brought together and be given a coherent and rational explanation.

Summary

The mechanism of corrosion of stainless steel 347 by fuming nitric acid has been determined to be approximately

$$2\ \overset{+}{NO_2} + Fe \rightarrow \overset{++}{Fe} + N_2O_4.$$

The rate can be described by the equation:

$$R = a\ (\overset{+}{NO_2})^{\frac{4}{3}} \quad e^{\frac{-\Delta h}{RT}}$$

and the constants *a* and Δh have been determined.

References

1. ROBERTSON, G. D., D. M. MASON & B. H. SAGE. 1952. Electrolytic conductance of the nitric acid-nitrogen dioxide-water system at 32°F. Ind. Eng. Chem. **44**: 2928.

2. INGOLD, C. K., E. D. HUGHES & R. I. REID. 1950. J. Chem. Soc. : 2400.

SECTION OF BIOLOGY

HISTOGENESIS OF THE *TU-E* MELANOMA IN *DROSOPHILA**

By Martin L. Kaplan

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The earliest reference in the literature to "tumors" in the Insecta is found in a treatise on insect diseases by Kirby and Spence in 1826.[1] Approximately 50 hereditary tumor-bearing stocks have been described in *Drosophila* since 1916 when Bridges[2] and Stark[3] first called attention to their presence in this genus. To date, the main emphasis in *Drosophila* tumor research has been on the penetrance of the genetic factors involved and on the influence of environmental factors. Comparatively few complete studies of tumor histogenesis have been forthcoming.[4,5,6] It is the purpose of this report to delineate the chronological development of the tumor found in the *tu-e* strain of *Drosophila melanogaster* and to demonstrate the evidence for the secondary invasion of normal tissues.

The relatively rapid formation of melanin in the tumors at 25° C. obscured cellular detail, except in the earliest stages, and made it difficult to describe adequately tumor ontogony. By exposing second instar larvae to 32° C. for 48 hours,[7] however, a retardation in pigment formation was obtained. Not only has the cellular picture emerged clearly, but the pattern of melanin deposition within the tumor has been sharply delineated.

Three phases in tumor histogenesis have been recognized in the *tu-e* genotype: (1) organization phase; (2) enlargement phase; and (3) melanization phase. The third has been subdivided into a peripheromelanotic stage and a centromelanotic stage. Six tumor sites have been found in the *tu e* larvae: pericardial cells; fat-body; tracheal tubes; imaginal discs; muscle; and hemolymph. The common denominator at all the observed tumor sites was the spindle-shaped cells which were first observed in the larval hemolymph at 72 hours of larval development. No spindle-shaped cells were seen in either of two nontumorous control stocks.

Organization Phase

Since the chief point of attack is the pericardial cells, a detailed

*This paper, illustrated with lantern slides, was the first of four papers in a symposium on Hereditary Melanotic Tumors in *Drosophila* presented at a meeting of the Section on January 10, 1955.

The work reported in the four papers comprising this symposium was supported in part by grants from the National Cancer Institute, National Institutes of Health, United States Public Health Service (C-1580), and in part by The Damon Runyon Memorial Fund for Cancer Research, Inc., New York, N. Y.

analysis of this tumor will be presented.

Between 72 and 78 hours of larval development, aggregations of spindle cells congregated around one or more pericardial cells. Spherical hemocytes were often included among the constituents. By 84 hours, the cell clusters had arranged themselves into a continuous strand around the pericardial cells and appeared to be increasing in number. The gradual accumulation of tumor cells plus the partial orientation of these cells was characteristic of this phase.

Enlargement Phase

The marked increase in pericardial tumor size began at 114 hours and reached its maximum at 120 hours. By this time, the tumor had approximately doubled its size and appeared as ovoid or teardrop-shaped when viewed in longitudinal section. In some cases, the long axis extended over one abdominal segment. The pericardial cells were now completely isolated from each other and from their position near the heart. The degree of compactness of the tumor depended on the make-up of the cellular constituents. The greater the number of spindle cells, the less compact it appeared. The intermingling of the filamentous ends gave a meshwork appearance to the tumor.

It was during this enlargement phase that the invasion of adjacent tissue, particularly fat-body and heart, occurred. In several 120 hour larvae, pericardial tumors were observed to contain at least one fat cell. In one case, for example, a tumor approximately 660 μ in length and applied to the ventral aspect of the heart, had protruded into, and replaced the fat-body in this region. Seven morphologically intact fat cells, as well as six intact pericardial cells, were incorporated within the body of the tumor. Penetration into the heart cavity by spindle cells from the primary tumor was also in evidence.

Melanization Phase

The onset of the melanization phase was clearly visible at 114 hours, thus overlapping the enlargement phase to some degree. At this time, isolated spindle cells, as well as spherical hemocytes located near the periphery of the tumor, took on a slightly yellowish tinge. Melanization always started at the cell periphery and progressed toward the cell center. The same pattern applied to the tumor as a whole, the melanized cells gradually linking up to form a more or less regular band enclosing a central amelanotic core. Tumors at this stage (120 to 126 hours) were designated as peripheromelanotic. Pericardial cells and fat cells never became melanotic. Instead, these cells underwent necrotic changes and were finally observed as irregular clumps surrounded by melanized spindle cells. Completely melanized pericardial tumors (centromelanotic stage) were never seen in the 32° C. shock series. Since centromelanotic

tumors were observed at other sites, however, it is possible that completion of the melanotic phase in pericardial tumors occurs after termination of the larval period. A summary of pericardial tumor histogenesis appears in FIGURE 1.

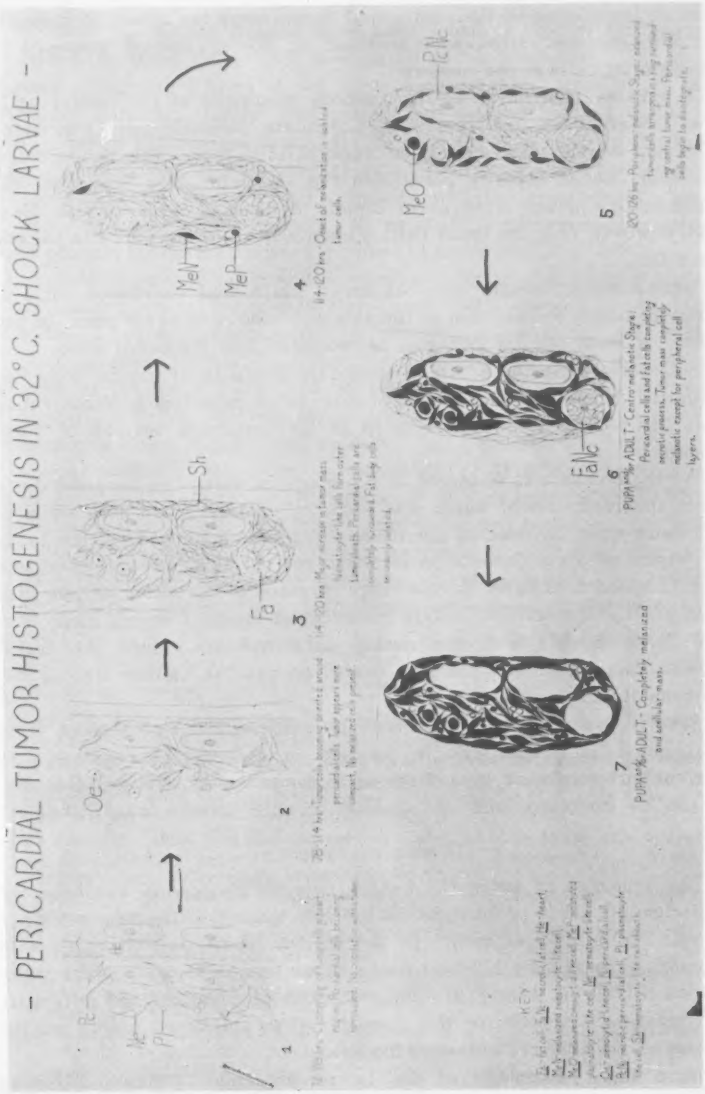

FIGURE 1

Tumors at Other Sites

Spherical clusters of spindle cells appeared in the hemolymph soon after the onset of pericardial tumor organization. These masses were observed to follow the sequence of events previously described. No pericardial cells were ever observed in these tumors. The possibility that at least some of the unattached tumors were the result of fragmentations at other sites was revealed by the occasional presence of necrotic fat cells in the masses.

A primary infiltration of the fat-body exclusive of pericardial cells was observed in *tu-e* larvae at 126 hours. Spindle cells penetrated between adjacent fat cells and encapsulated them. The tumor cells, however, never actually penetrated the fat cells. The stages of the melanization phase were never clearly defined in these tumors. Here too, however, only the tumor cells melanized, while the fat cells became necrotic.

Occasionally, tumors were observed which had enveloped parts of tracheal tubes. Penetration of the target tissue was never seen. In one case, at least one fat cell was incorporated into the tumor mass. This would seem to indicate that a secondary invasion of the fat-body had occurred, since only one tumor of this nature was found. Melanization was limited to a few tumor cells in the peripheral regions of these tumors.

A single case was observed at 126 hours in which two imaginal discs were involved, one of which was probably destroyed. No pericardial or fat cells were included in the tumor mass. The main body of the tumor consisted of a conglomeration of melanized cells, spherical hemocytes, and imaginal disc cells. A secondary invasion of the intact haltare disc was clearly discernible. In this instance, at least, it seems likely that the larva would have died during metamorphosis, since the dorsal mesothoracic disc was probably destroyed and the haltare disc in the process of being obliterated.

Several instances were observed in which a mass of spindle cells surrounded individual muscle bundles in the caudal hemocoel. No pericardial cells were ever observed in these tumors. Melanization was slight to moderate, and no apparent muscle damage was discerned.

Concluding Remarks

The presence of *melanotic* masses in adult *Drosophila* has been the criterion for tumor penetrance. In the *tu e* genotype, the adults have a penetrance of 96 per cent.[7] In the current report, the observation of amelanotic tumors in 126-hour nontumorous *tu-e* larvae raises the penetrance to 100 per cent. This datum, therefore, questions the reliability of penetrance studies in the literature that are based solely on the presence of melanized tumors in the *adult*.

In a high percentage of the larvae examined, pericardial tumors

appeared prior to tumor formation at other specific sites. Hamly[8] has demonstrated the presence of a tumor agent in the *tu-e* larvae. It may well be that the invasion of target tissue in this genotype is a function of the threshold level of tumor agent coupled with tissue reactivity to the agent.

To date, no mitotic figures have been found in any of the tumors described in *Drosophila*. The *tu-e* strain is no exception. The mere absence of mitotic figures at the time of observation, however, is not sufficient criterion. Harnly[9] has pointed out that he has yet to see a mitotic figure in imaginal discs. It is quite possible that, in the *tu-e* tumor, a relatively short period of rapid proliferation exists between 114 to 120 hours. This interval coincides with the enlargement phase of tumor ontogeny. It is difficult to conclude that aggregation alone could account for the tremendous increase in tumor size.

It has been pointed out that the spindle cells are the common factor at all tumor sites in the *tu-e* strain. These cells were never observed prior to 72 hours of development. It seems likely that the secondary invasion of other tissues from the primary sites represents metastatic behavior of the tumor cells. The envelopment and subsequent necrosis of pericardial cells, fat-body cells, and imaginal disc cells indicates a destructive effect of tumor cell activity.

Several problems raised as an outgrowth of this study are under investigation: (1) What is the ultimate origin of the tumor cells? (2) What factors are responsible for the induction of tumor cell formation? (3) What role do the tumor cells play in the necrosis of infiltrated tissues? (4) What factors are involved in the onset of melanization in the tumor? (5) What factors govern the specific pattern of melanin formation in the tumors?

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SECTION OF BIOLOGY

EFFECTS OF VITAMINS AND THEIR ANALOGS UPON TUMOR INCIDENCE IN *DROSOPHILA MELANOGASTER**

By Frank Friedman**

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Reports of nutritional variations and modified adult melanotic tumor incidence in various strains of *Drosophila melanogaster* indicated that the yeast component in the diet of this insect was critical.[1,2,4,6,8] Preliminary tests upon our *tu-e* stock revealed that the substitution of a polyvitamin compound for the "live" yeast component in an otherwise normal diet simulated the procarcinogenic effects of the "live" yeast factor.[3] The tumor penetrance of the *tu-e* strain (number of adults displaying the pigmented growth) under optimum conditions of diet and temperature (25±0.5° C.) ranged from 80 to 85 per cent.[3] Of every 100 eggs laid, 70±5 emerged as adults 9.3 days after egg laying. The appearance of the pigmented growth in this ebony body color, scarlet eye strain of *Drosophila* is dependent upon a recessive gene located on the right arm of the second chromosome at locus 88.[5]

In order to control the amount of yeast available to the developing animals, "dead" brewer's yeast*** (yeast subjected to a steam pressure system to kill all spores) in an agar-water base was used as a test food. Thus the vitamin content of the diet could be ascertained and sterile conditions could be maintained. In all the foregoing tests, larvae were introduced, using sterile techniques, into experimental vials in groups of 15 and were incubated at 25±0.5° C.

When the concentration of "dead" yeast was reduced from 15 to 2.5 per cent, the tumor incidence of animals reared on these diets fell from 80.3 per cent to 25.9 per cent (TABLE 1). Despite the reduction in tumor incidence, there was not a significant change in survival. The developmental period of animals reared on the lower yeast medium, however, was prolonged to 10 days (TABLE 1). These results substantiated a previous contention that animals developing in a culture that had been depleted of "live" yeast displayed a low tumor incidence.[3]

*This paper, illustrated with lantern slides, was the second of four papers in a symposium on Hereditary Melanotic Tumors in *Drosophila* presented at a meeting of the Section on January 10, 1955.

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***Supplied by Standard Brands, Inc., Fleischmann Yeast Co., Pearl River, N. Y.

Amount of vitamin per gram of dead brewer's yeast (in micrograms per gram of yeast)

Thiamine	10.0	Pyridoxine	40.0
Riboflavin	70.0	Paraminobenzoic acid	5.0
Niacin	500.0	Inositol	4000.0
Pantothenic acid	150.0	Choline	4000.0
Biotin	1 to 2.0	Ergosterol	9000.0
		Folic acid	25.0

TABLE 1

EFFECTS OF "DEAD" YEAST CONCENTRATION UPON TUMOR INCIDENCE, SURVIVAL, AND DEVELOPMENTAL TIME

Per cent "dead" yeast	*Larvae tested*	*Imagos*	*Tumorous imagos*	*Penetrance*	*Per cent survival*	*Developmental period in days*
15.0	1140	709	569	80.3	62.2	9.3
2.5	960	580	150	25.9	60.4	10.0
Normal diet	1170	786	654	83.2	67.2	9.3

TABLE 2

RESULTS OF INDIVIDUAL AND MULTIPLE ADDITIONS OF VITAMINS* TO A LOW TUMOR MEDIUM

Vitamin	*Larvae tested*	*Imagos*	*Tumorous imagos*	*Penetrance*	*Per cent survival*	*Days to emerge*
T	1065	684	298	43.6	64.2	9.3
R	930	574	255	44.4	61.7	9.3
P	1140	718	339	47.2	62.9	9.3
B	1155	748	436	58.3	66.7	9.3
N	1000	611	377	61.7	61.0	9.3
NT	1170	704	349	43.4	60.3	9.3
NR	1095	686	357	52.0	62.7	9.3
PR	1080	694	315	45.4	64.3	9.3
PT	1125	733	347	47.3	65.2	9.3
PN	1155	781	369	47.2	67.6	9.3
BN	1170	813	472	58.1	69.5	9.3
BR	1140	775	415	53.5	67.9	9.3
BT	1125	739	375	50.7	65.6	9.3
BP	1155	833	517	62.1	72.1	9.3
TR	1065	684	325	47.5	64.2	9.3
NRT	1200	837	427	51.2	69.8	9.3
PNR	1125	759	375	49.4	67.5	9.3
PNT	1110	746	344	46.1	67.2	9.3
PRT	1170	870	369	45.7	68.9	9.3
BNT	1185	848	431	50.8	71.6	9.3
BNR	1170	841	417	49.6	71.9	9.3
BRT	1110	705	328	46.5	63.5	9.3
BPT	1170	812	479	58.9	69.4	9.3
BPN	1155	862	539	62.5	74.6	9.3
PBR	1095	716	439	61.3	63.7	9.3
PRTN	1125	700	326	45.1	62.2	9.3
BRTN	1140	740	343	46.4	64.9	9.3
BPRT	1155	792	487	61.5	68.6	9.3
BPRN	1110	771	437	56.7	69.5	9.3
BPTN	1125	781	408	52.2	69.4	9.3
BPRTN	1185	867	543	62.6	73.2	9.3

*N = Niacin
B = Biotin
P = Sodium Pantothenate
R = Riboflavin
T = Thiamine Hydrochloride

When reared on a normal diet of cornmeal-molasses seeded with "live" yeast *or* on a 15 per cent "dead" brewer's yeast medium, animals of the *tu-e* stock displayed similar tumor frequencies, survival values, and developmental periods (TABLE 1). Therefore, by ascertaining the difference in vitamin content between the high tumor medium (15 per cent "dead" brewer's yeast) and the low tumor medium (2.5 per cent "dead" brewer's yeast), it was possible to determine the quantity of vitamin that need be added to the low tumor medium. We postulated that the addition of vitamins to the low tumor medium would increase the tumor incidence of animals reared thereon and thus indicate the vital role of vitamins in tumor gene expression.

The results, as tabulated in TABLE 2, demonstrate that in each case the tumor incidence of animals raised on a vitamin-supplemented medium was notably higher than that of animals reared on a low tumor medium alone (TABLE 1). The developmental period for experimental animals returned to 9.3 days and their survival equalled or surpassed that of animals developing on the low-tumor medium alone (TABLES 1 and 2). Those vitamin additions which most enhanced the adult expression of the tumor gene were: the individual additions of biotin *or* niacin; the dual additions of biotin with niacin *or* sodium pantothenate;

TABLE 3

VITAMIN SUPPLEMENTED LOW TUMOR MEDIUM PLUS VARIOUS CONCENTRATIONS OF CORRESPONDING VITAMIN ANALOGS

Vitamin plus concentration of analog	*Larvae tested*	*Imagos*	*Tumorous imagos*	*Penetrance*	*Per cent survival*	*Days to emerge*
Riboflavin plus lumiflavin						
Equal amts.	870	570	344	60.4	66.8	9.3
10x	900	561	422	75.2	62.3	9.3
50x	855	423	182	43.0	49.5	9.3
Na pantothenate plus pantoyltaurine						
Equal amts.	795	510	221	43.4	64.2	9.3
10x	870	544	412	75.8	62.6	9.3
50x	840	476	190	39.9	56.7	9.3
Thiamine HC1 plus pyrithiamine						
Equal amts.	810	514	173	33.7	63.5	9.5
10x	840	530	148	27.9	63.1	9.5
50x	870	232	60	25.9	26.7	9.5-6
Biotin plus desthiobiotin						
Equal amts.	780	461	207	44.5	59.1	9.5
10x	870	571	248	43.4	65.6	9.5
50x	900	568	240	42.3	63.1	9.5

the triple additions of biotin and sodium pantothenate with thiamine hydrochloride, niacin, or riboflavin; the quartet supplement of biotin, sodium pantothenate, riboflavin, and thiamine hydrochloride; and the quintet supplement of all five vitamins.

In order to corroborate these results, analogs of the foregoing vitamins were incorporated in the vitamin re-enforced low tumor medium. Each analog was added with its vitamin in amounts "equal to," tenfold, or fiftyfold the quantity of supplemented vitamin. The analogs employed were lumiflavin (for riboflavin), pyrithiamine (for thiamine hydrochloride), pantoyltaurine (for sodium pantothenate), desthiobiotin (for biotin), and four analogs of niacin, *i.e.*, 3-acetyl pyridine, pyridine-3-sulfonic acid, isonicotinyl hydrazide, and picolinic acid.

The tumor incidence of animals reared on a riboflavin supplemented low-tumor medium was 44.4 per cent (TABLE 2). This value was not significantly reduced with the addition of increasing concentrations of lumiflavin (TABLE 3). The developmental period of animals raised on all concentrations of this analog was 9.3 days, and no lethal effects were apparent until the analog concentration was increased to fiftyfold. Contrary to expectation, tumor incidence *increased* with the two lower concentrations of this analog.

The effects of pantoyltaurine were similar to those described for lumiflavin as affecting tumor incidence, survival, and the developmental period (TABLE 3).

Animals raised on a thiamine hydrochloride-supplemented low tumor medium displayed a tumor incidence of 43.6 per cent, survival of 64.2 per cent, and a normal developmental period for this stock, 9.3 days (TABLE 2). At the "equal" concentration, pyrithiamine reduced the tumor frequency of animals reared on the thiamine hydrochloride-augmented low tumor medium by about one third, increased the developmental period by four hours, but did not alter survival (TABLE 3). At the tenfold concentration of analog, the procarcinogenic effects of thiamine hydrochloride were virtually eliminated without affecting survival. The developmental period was prolonged to 9.5 days (TABLE 3). At the fiftyfold concentration of pyrithiamine, the procarcinogenic effects of thiamine hydrochloride were again canceled, but with severe mortality amongst the developing animals. The developmental period was not significantly increased above the afore-mentioned time of 9.5 days.

Regardless of desthiobiotin concentration in a biotin-augmented low tumor medium, the procarcinogenic effect of biotin (58.3 per cent, TABLE 2) was reduced approximately 27 per cent (TABLE 3). Survival of animals on the biotin-desthiobiotin media was not significantly different from that of animals raised on the biotin re-enforced low tumor medium alone (TABLES 2 and 3). The developmental period of animals reared on all desthiobiotin biotin media was 9.5 days.

The effects of niacin analogs are given in TABLE 4. Of the four

niacin analogs employed, isonicotinyl hydrazide and α picolinic acid were able to reduce the procarcinogenic effect of niacin (penetrance-61.7 per cent, TABLE 2). Pyridine-3-sulfonic acid had no adverse effects upon tumor incidence, and 3-acetyl pyridine canceled the procarcinogenic effect of niacin, but with an accompanying severe mortality (TABLE 4). The survival of animals raised on a niacin-augmented low tumor medium was 61.0 per cent, and the developmental period was 9.3 days (TABLE 2). The survival of animals reared on media containing successful niacin analogs did not vary from the foregoing survival value, but the developmental period was prolonged to 9.5 days (TABLE 4). At both the "equal" and tenfold concentrations of α picolinic acid, the niacin procarcinogenic effect was almost halved (TABLE 4). As the concentration of isonicotinyl hydrazide was increased from "equal" to a tenfold level, the procarcinogenic effect of niacin was reduced to 46.7 and 37.4 per cent respectively (TABLE 4). At a fiftyfold concentration of α picolinic acid, isonicotinyl hydrazide, and 3-acetyl pyridine larval death ensued.

In the foregoing tests it is seen that the adult expression of the tumor gene in this *tu-e* strain is closely associated with the diet of the animal. Herskowitz and Burdette[6] suggested that the "quantity and quality" of yeast in the diet of a tumor stock is critical in the expression of the gene-controlled melanotic growth found in *Drosophila melano-*

TABLE 4

NIACIN SUPPLEMENTED LOW TUMOR MEDIUM PLUS VARIOUS CONCENTRATIONS OF FOUR NIACIN ANALOGS

Analog	*Larvae tested*	*Imagos*	*Tumorous imagos*	*Penetrance*	*Per cent survival*	*Days to emerge*
Picolinic acid						
Equal amts.	855	551	195	35.4	64.4	9.5
10x	870	605	219	36.2	69.5	9.5
50x			Larval death			
Isonicotinyl hydrazide						
Equal amts.	870	553	258	46.7	63.5	9.5
10x	840	514	192	37.4	61.2	9.5
50x			Larval death			
Pyridine-3-sulfonic acid						
Equal amts.	840	462	232	50.2	54.5	9.3
10x	900	613	356	58.1	68.1	9.3
50x	870	567	336	59.2	65.2	9.3
3-Acetyl pyridine						
Equal amts.	870	502	320	63.7	57.7	9.3
10x	600	226	55	24.2	37.7	11.0
50x			Larval death			

gaster. Mittler[7,8,9] demonstrated that the type of yeast, amino acids, and vitamins in the diets of *Drosophila* tumor strains can alter the expression of adult tumor frequencies. It has been shown in these tests that vitamins are at least one of yeast nutrients intimately concerned with tumor penetrance in the *tu-e* stock. Since the usual tumor incidence for this strain (80 to 85 per cent) could not be attained by the use of vitamin-supplemented low tumor media, it appears that other vitamins and/or supplementary nutrients are necessary. Contrary to the results of the role of vitamins in spontaneous tumorous vertebrates,[10] the effects upon *Drosophila* tumor incidence appear to be independent of survival.

The use of the vitamin analogs seems to verify the close association between lowered tumor incidence and prolonged larval development. The inhibitory action of the successful analogs upon the procarcinogenic effect of their vitamin counterparts indicates a possible therapeutic value for these substances in neoplastic research. Current tests are centered about the effects of these analogs upon the carcinogenic activity of an agent extracted from larvae of the *tu-e* stock. These tests indicate that some of these analogs can nullify the carcinogenic activity of this agent.

In summary, it has been demonstrated that larvae of a *tu-e* strain of *Drosophila melanogaster*, which have an adult tumor incidence of 80 to 85 per cent, emerge with a tumor frequency of only 25.9 per cent when reared on a low tumor medium (2.5 per cent "dead" brewer's yeast). This decline is accompanied by a lengthened egg-larval developmental period. Animals reared on all low tumor vitamin-augmented low tumor media display a significantly higher tumor incidence. Certain individual and multiple vitamin supplements, however, are more effective than others in elevating tumor frequency. Of the analogs employed, lumiflavin, pantoyltaurine, and pyridine-3-sulfonic acid are not considered reliable antagonists of the procarcinogenic effects of their corresponding vitamins. It has been demonstrated that when vitamin analogs reduce tumor incidence, a concomitant lengthening of the animal's developmental period ensues. There does not seem to be a relation, however, between the latter period and survival to adulthood.

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SECTION OF BIOLOGY

CARCINOGENIC EFFECTS OF AN EXTRACTABLE LARVAL TUMOR AGENT*

By Lawrence Burton**

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Research has associated the formation of melanotic tumors in *Drosophila melanogaster* with tumor genes. The etiology of these tumors has been quite enigmatic. Harnly, Burton, Friedman, and Kopac (1954), however, have associated the formation of melanotic tumors in the *tu-e* strain with an etiological agent. This tumor agent, present in acellular larval extracts of the *tu e* strain, when injected into similar age larvae of two nontumorous strains, induced melanotic tumor formation. Although tumor agents have been demonstrated in two other tumor strains, they have not been found in extracts of three tumor-free strains. This investigation was conducted to elucidate the carcinogenic action of the *tu-e* tumor agent.

Two strains, *wild 51-52* and *tu-e* were utilized in this investigation. The *wild 51-52* is a genetically stable and hardy strain. Moreover, this strain has proven to be tumor-free, and larval extracts of this strain do not possess an active tumor agent (Harnly, Burton, Friedman, and Kopac, 1954). Nevertheless, this strain is highly reactive to the carcinogenic action of the *tu-e* tumor agent. The *tu-e* strain is a genetically stable but less hardy strain.

Histological sections of the induced tumors indicated that the induced tumors were similar to the tumors present in the *tu-e* strain.

Nine different larval donors and hosts were utilized in all age combinations. Extracts of 36-hour *tu-e* larvae also were injected into 40- to 72-hour hosts. Moreover, pupae (all ages) and adults of both strains were utilized.

Studies were conducted to ascertain the amount of fluid that could be obtained from donors at various stages of development. The concentration of larvae to eight drops of Waddington's salt solution*** was computed upon the concentration of the 120-hour extracts (FIGURE 1).

The larval extracts were prepared by placing the proper number of washed larvae into a mortar and grinding them into an amorphous mash, to which eight drops of Waddington's solution were added. This mixture was centrifuged at 5400 × g. for 15 minutes. Upon completion of the centrifugation, the supernatant fluid was pipetted into a glass tube.

*This paper, illustrated with lantern slides, was the third of four papers in a symposium on Hereditary Melanotic Tumors in *Drosophila* presented at a meeting of the Section on January 10, 1955.

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***Waddington's salt solution: NaCl 7.5 gm., $NaHCO_3$ 0.2 gm., $CaCl_2$ 0.2 gm., KCl 0.1 gm., distilled H_2O 1000 ml.

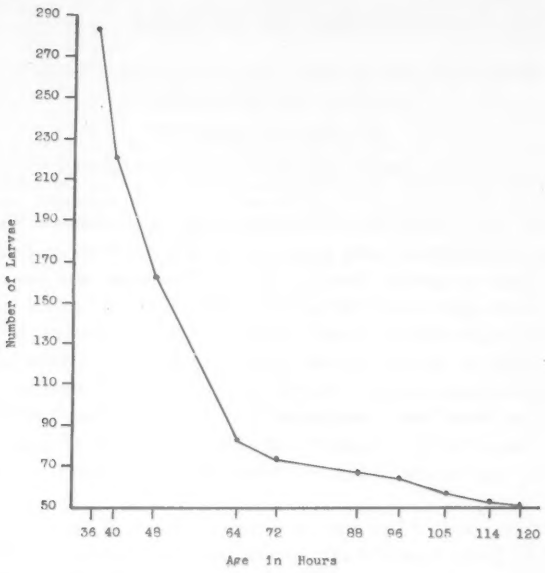

FIGURE 1. Compensated concentrations (larvae/ringer).

When the donors were either 114-hours or 120-hours, the supernatant fluid was transferred into a cooled glass tube which was placed in an ice bath. The ice bath was employed to delay a blackening of the fluid that invariably occurred. (The blackened fluid proved to be so toxic that no host larva injected with it ever survived the operation).

The acellular nature of the supernatant fluid was regularly tested microscopically. These tests indicated that all cells and debris were sedimented.

After the extracts had been prepared, the washed hosts were placed upon a slide and etherized. After etherization, the larvae were dried and aligned upon the slide.

The injection apparatus was essentially similar to that described by Ephrussi and Beadle (1936), except that more finely pointed pipettes (diameter 0.002 to 0.015 mm.) were utilized. After the fluid extract was drawn into the pipette, the pipette was inserted into the larva at the third or fourth posterior segment. The pipette, inserted almost parallel to the exoskeleton, was not permitted to approach any of the deeper internal organs. Mere insertion of the pipette did not produce melanotic tumors.

Waddington's solution was injected into larval hosts (all ages), and none of the 357 surviving hosts developed tumors.

Extract fluid was injected until each larva was slightly distended.

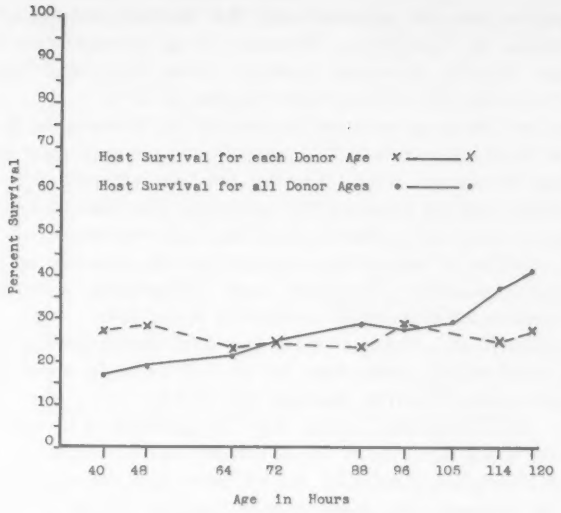

FIGURE 2. Mean postoperative survival.

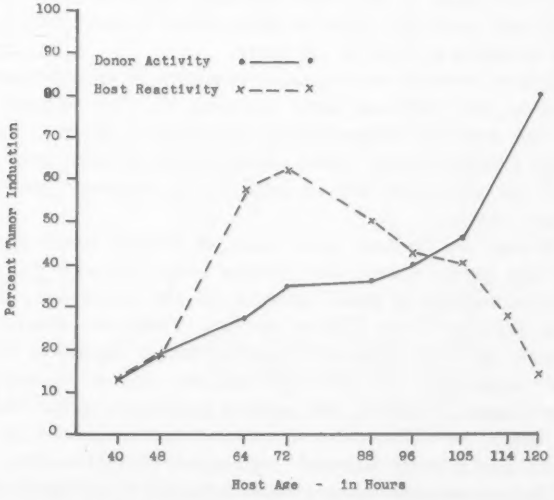

FIGURE 3. Mean donor activity and host reactivity.

The pipette was not removed until the internal pressure of the larva was visibly at equilibrium. Recovery of all postoperative hosts in a moisture chamber increased survival. After transferral into vials of food, the hosts continued their development at 25° C.

Only two donor-age extracts possessed the tendency to blacken. The rate of blackening of the 120-hour extracts occurred more rapidly, and in fewer observable steps, than the 114-hour extracts. All other donor-age extracts did not evidence this tendency. The other extracts, however, did appear quite turbid after 24 hours at room temperature.

No reduction of extract carcinogenic activity occurred up to 10 hours following preparation. Moreover, some carcinogenic activity has been maintained in the extracts for a period of seven days.

Injection of *tu-e* larval extracts into host larvae usually retarded the development of the hosts from two to five days. In some instances, a few hosts never achieved pupation and died as larvae after two to three weeks. The retardation factor may be attributed to only *tu-e* larval extracts, since injection of larval extracts of two tumor-free strains, as well as Waddington's solution, did not retard host development.

In no instance did postoperative survival significantly affect the observed incidence of induced melanotic tumors (FIGURE 2). Survival increased with increased host age.

The injection of 36-hour donor extracts into the 40- to 72-hour hosts demonstrated the presence of a weak carcinogenic agent.

Mean carcinogenic activity of the donor extracts rose from a low at 40 hours to a peak at 120 hours (FIGURE 3). In contrast, mean host reactivity also rose from a low at 40 hours to a peak at 72 hours, but declined thereafter to a low at 120 hours.

The 40-hour extracts induced tumor formation in all age hosts except the 114-hour and 120-hour hosts (FIGURE 4). The 48-hour, 64-hour, and 72-hour extracts induced tumor formation in all host age groups except the 120-hour hosts. These curves appear to differ only in magnitude, and the differences may be attributed to increased concentrations of the tumor agent.

The 88-hour and 96-hour donor extracts induced tumor formation in all host age groups except the 120-hour hosts (FIGURE 5). The mean carcinogenic activity of these extracts did not statistically differ from the mean activity of the 72-hour extracts. These two curves, from 40 to 72 hours, possibly represent tumor incidences similar to the 72-hour extracts' incidences, and this may indicate similar concentrations of the tumor agent. However, the greater incidence in the 88-hour and 96-hour hosts, as well as the sharply reduced incidences in the 114-hour hosts, may indicate different carcinogenic characteristics.

The 114-hour extracts induced tumor formation in all age hosts except the 40-hour and 48-hour hosts (FIGURE 6). The lack of carcinogenic activity of the 114-hour extracts in the 40-hour and 48-hour hosts, as

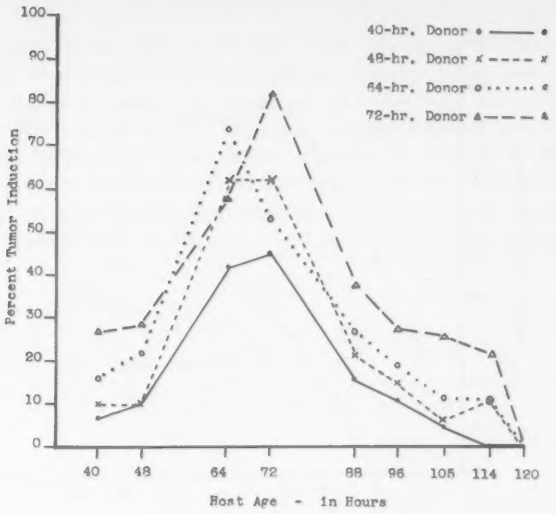

FIGURE 4. Donor carcinogenic activity (Agent *A*).

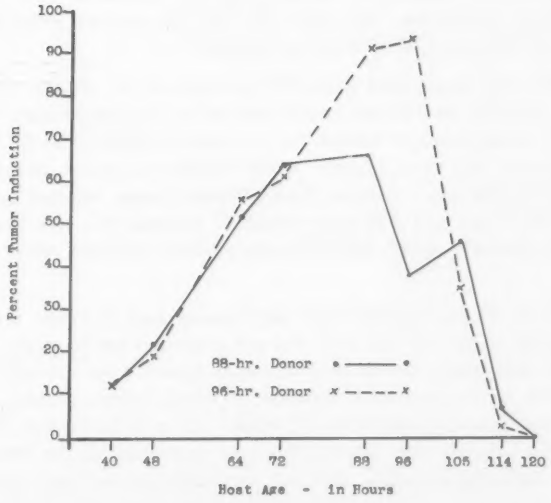

FIGURE 5. Donor carcinogenic activity (Agent *B*).

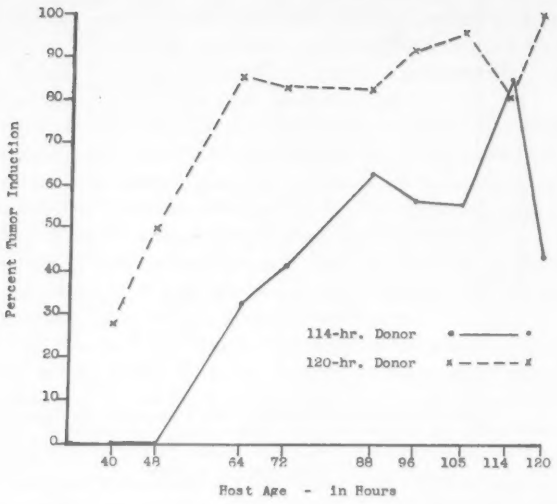

FIGURE 6. Donor carcinogenic activity (Agent C)

well as their positive activity in the 120-hour hosts, may indicate the acquisition of new carcinogenic activity. The 120-hour donor extract inductivity curve possibly indicates greatly increased and different carcinogenic properties. All host age groups reacted greatly to the carcinogenic effects of this donor-age extract.

Different age hosts had different reactances to various donor-age extracts. The 40- to 72-hour hosts reacted to the carcinogenic properties of all donor extracts except the 114-hour extracts. The 88-hour and 96-hour hosts had their highest tumor incidences when injected with 96-hour and 120-hour extracts. The 114-hour hosts reacted greatly to only the 114-hour and 120-hour extracts, whereas the 120-hour hosts had their greatest incidence of tumors when injected with 120-hour extracts.

Extracts of 96-hour larvae were overconcentrated 16 times the normal concentration of larvae and still did not evidence carcinogenic activity in the 120-hour hosts. Extracts of 120-hour donors were diluted 30 times the dilution of Waddington's solution to donor larvae. These extracts still induced tumor formation in all of the 120-hour hosts tested. These experiments indicate that the carcinogenic properties of the 96-hour and 120-hour extracts possibly differed qualitatively as well as quantitatively.

A series of re-extraction experiments were conducted which consisted

of the injection of an extract into hosts, the subsequent re-extraction of these hosts after a specific period, and the injection of the new extracts into other hosts (FIGURE 7). Injection, re-extraction, and reinjection of 72-hour extracts indicated that the 72-hour extracts acquired the carcinogenic properties of the 96-hour extracts at from 72 to 96 hours of host age, acquiring the characteristic activity of the 120-hour extracts at from 72 to 120-hours of host development. Injection of 96-hour extracts, and subsequent re-extraction and reinjection, indicated that the

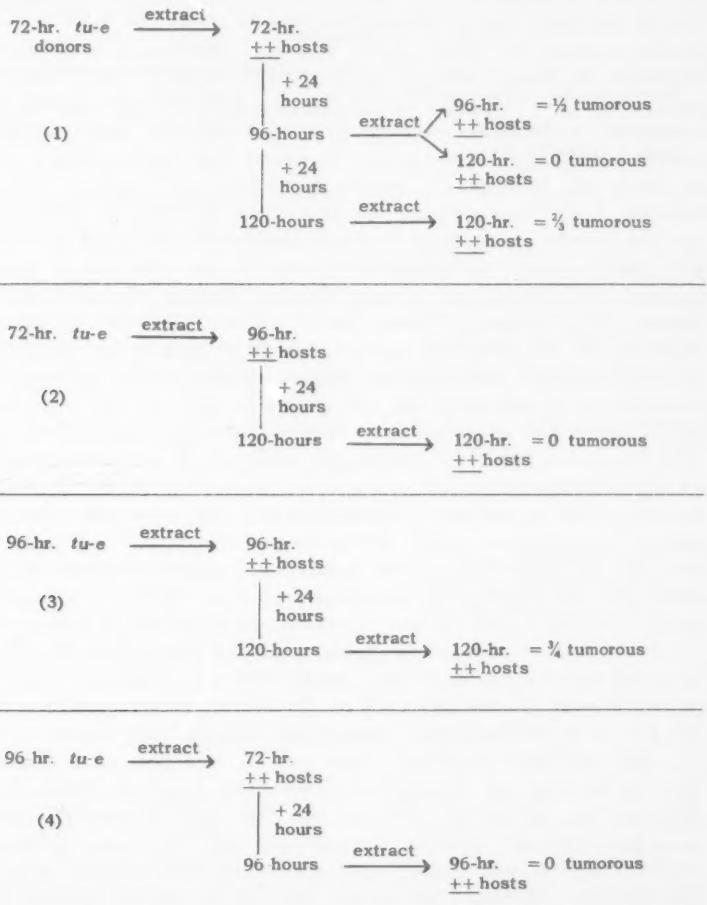

FIGURE 7. Results of re-extraction experiments.

96-hour extracts only acquired the characteristic carcinogenic activity of the 120-hour extracts at from 96 to 120 hours of host development.

The injection of 96-hour, 120-hour, pupal, and adult extracts into pupal and adult hosts indicated that pupal and adult hosts were not competent to react to the tumor agent. Moreover, since *tu-e* pupal and adult extracts did not induce tumor formation in the 64- to 120-hour hosts, an active tumor agent may be present only in larval extracts. Therefore, this tumor agent is a larval tissue derivative, and its activity is larval tissue specific.

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SECTION OF BIOLOGY

THE *TU-E* GENE ACTION AND TUMOR DEVELOPMENT*

By Morris Henry Hamly

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The appearance of the *tu-e* tumor in individuals of this strain of *Drosophila melanogaster* is dependent upon the presence of a recessive gene at locus 88± in the second chromosome. Doctor Burton has demonstrated by the injection of the *tu-e* larval extract that, given a minimal quantity of the gene-determined carcinogen, other genotypes containing the normal allele are also capable of tumor development. It is evident that this tumor is, in part, determined by the interaction of the *tu-e* gene's product with the products of the rest of the genotype—a physiological gene-products interaction system. In addition, the data on temperature, provided by Mrs. Emery, and the data on vitamins, provided by Doctor Friedman, demonstrate that both external and internal environmental factors enter into or affect this interaction system. Finally, the X-irradiation work of Miss Fleischer suggests that possibly the threshold and the competency of specific larval tissues to respond to this genetic carcinogen are factors in tumor formation. The presence of this tumor is dependent upon a complex genetic, environmental, and developmental interaction system. What role does the *tu-e* gene play in this system?

A partial and suggestive answer is possible at this stage of the investigation. The 36-hour (from egg-laying) *tu-e* larval extract-inducing tumors in the 51 to 52 wild, nontumorous strain demonstrates that by this time the tumor gene has synthesized a carcinogen which, even in the presence of the product of the normal allele, is capable of entering into the host physiological interactions and so shift or alter the direction of these reactions that tumors are produced. The curves of donor extracts (see graphs in Doctor Burton's report) from 40- to 72-hour *tu-e* larvae into the 40- to 120-hour aged hosts are all bell-shaped distribution curves skewed to the left to the same degree, and constitute a single family of curves. These curves differ only in the magnitude of tumor-inductive power, hence they measure increasing concentrations of a common carcinogen we may call *A*. One could explain the positive slope of these curves as due to more units of *A* injected per larva as the host larvae increase in size with age, but this explanation will not hold beyond 72 hours, when the slope becomes negative, but still more units of *A* are injected into these much larger larvae. The common positive and negative slopes of the four curves may, possibly, measure the

*This paper, illustrated with lantern slides, was the fourth of four papers in a symposium on Hereditary Melanotic Tumors in *Drosophila* presented at a meeting of the Section on January 10, 1955.

reaction threshold or competency changes with age of the host larval tissues to carcinogen *A*. The low tumor frequency found in 40- and 48-hour hosts would suggest that either too few units were injected, due to the very small size of these larvae, or that the injected agent *A* was metabolized before the host tissue had reached the labile or reactive stage in ontogeny, the occasional induction in 40- and 48-hour hosts being due to individuals more advanced physiologically.

The donor extracts from 88- and 96-hour *tu-e* larvae produce tumor frequencies in hosts of 40 to 72 hours of age similar to the incidence from donors of 40 to 72 hours of age, and therefore show the presence of agent *A* in the 88- and 96-hour extracts. But the tumor frequency is much higher in hosts aged 88 and 96 hours. This frequency variance indicates something different in the extracts from 88- and 96-hour donors not present in extracts from younger donors. Those extracts containing both entities *A* and *B* produce a low tumor frequency in 105- and 114-hour hosts and fail to induce tumors in 120-hour hosts. The concentration experiments of Doctor Burton, with the 96-hour donor extract and dilution of the 120-hour donor extract, indicate the presence of a third agent *C* in the 120-hour extract.

If *B* is not *C*, then 105-hour host tissue has either a very high threshold reaction to *A*, an intermediate threshold to *B*, and a very low threshold for *C* or else (considering the short time the agent has to act in these 105-hour-late larvae before puparium formation and lysis of the larval tissues begins), the entities *A*, *B*, and *C* represent steps in the activation of the carcinogenic agent to more and more potent forms of the carcinogen. For various reasons, we believe that *B* may be a transitory entity in the conversion of *A* to *C*. It appears to be highly labile going to *C*, or is utilized in a noncarcinogenic manner.

Since the 96-hour donor extract curve in 40- to 72-hour hosts is an agent *A* curve, then this is evidence that the *tu-e* gene, having begun to produce the carcinogen *A* at some moment before 36 hours of development and having brought it to maximum concentration, continues to produce *A* and maintain it at this concentration until at least 96 hours of total development. Since the 114-hour donor extract does not induce melanotic tumors in 40- and 48-hour host larvae, this extract must be an *A*-free extract. Between 96 and 114 hours the *tu-e* gene stops producing *A*, and all of it has either been converted to *B* or metabolized. Furthermore, host tissues of 40- and 48-hour development are not responsive to *B* and, by the time they are at a developmental stage when they could respond, *B* has either been metabolized by the host or eliminated from the body.

The 88-hour donor curve demonstrates, by its higher values at 88- to 105-hour host reaction, that *B* must have begun to appear for the first time in the *tu-e* animal some time between 72 and 88 hours of development.

As demonstrated above, the entities *A* and *B* exist as different entities appearing successively but overlapping in time. The concentration and dilution experiments demonstrate these entities will not induce tumors in 120-hour hosts. No entity exists as late in development as the 105-hour donor extract that will induce tumors in the 120-hour host. But the injections of extracts from 114- and 120-hour donors do induce melanotic tumors in 120-hour hosts. Therefore, these tumors must have been induced by a third entity developed in time, carcinogen *C*, and this must first appear between 105 and 114 hours of development.

The 114 hour donor curve proves the absence of the carcinogen *A*, since it does not induce tumors in 40- and 48-hour hosts, and demonstrates the presence of a different carcinogen *C*, since it does induce tumors in 120-hour hosts. This curve may be a mixed curve for agents *B* and *C*. Since the 114-hour donor curve demonstrates both the absence of *A* and the inability of 40- and 48-hour hosts to react to *B*, then the 120-hour donor curve at 40- and 48-hour hosts is a pure *C* curve.

Summary

Beginning at some moment following fertilization, the *tu-e* gene initiates an activity resulting in the production by 36 hours of development of an extractable carcinogen and maintains its presence in high concentration for 60 hours (present in 96-hour donor larvae). This effect, at the present stage of the investigation, is the only one of the *tu-e* gene of which we can be certain, since, in the injection, re-extraction, and reinjection experiments the nontumorous genotype host can carry *A* to *C*. At some point beyond 96 hours of development, this *tu-e* gene, having initiated and maintained the reaction producing *A*, ceases, and no trace of the carcinogen *A* is detectable then in 114-hour donor *tu-e* larvae, carcinogen *A* having been either metabolized or converted by way of *B* to *C* in the *tu-e* larvae. Extracts from the nontumorous genotype larvae demonstrate that the normal allele does not initiate this reaction producing the carcinogen *A*, nor is *C* ever present. Given the carcinogen *A* by injection, re-extraction experiments demonstrate that one can recover the carcinogen *C* from the host. At present, there are at least two possible explanations for the origin of the carcinogen *C*. Either the carcinogen *A* enters into the system of physiological reactions, controlled by the common gene complex of both the tumorous donor and of the nontumorous host strains, and shunts it into a different course, producing, by way of *B*, the carcinogen *C*, or the entity *A* may not be related to *C* at all. *A* may be the *tu-e* gene-initiated carcinogen responsible for the initial conversion of normal cells to tumor cells. The carcinogen *C* may then be one of the products of tumor metabolism. Since the carcinogen *C* is not detectable until late in ontogeny (114- and 120-hour donor larvae), by which time the tumor is fully formed and large in the donor, one is at present inclined toward the latter interpretation.

SECTION OF PSYCHOLOGY

THE HISTORICAL MEANING OF PSYCHOLOGY AS A SCIENCE AND PROFESSION*

By Rollo May

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Psychology, until about 100 years ago, was part of philosophy and ethics. The great psychological insights before the time of Freud are to be found in the writings of philosophers and religious teachers such as Socrates, Plato, Augustine, Pascal, Spinoza, Kierkegaard, and Nietzsche. The first emergence of psychology in the experimental scientific sense was relatively late; Wundt established his psychological laboratory in Leipzig in 1879 for the exploration of physiological psychology.

To understand the meaning and development of contemporary psychology, we need to see briefly what has happened in man's relation to nature and to himself in the modern period. Since the Renaissance, as is well well known, a new view of the objectivity of man arose. For complex reasons that we shall not discuss here, natural science in its modern mathematical form was founded on a career to be marked by tremendous progress. When Descartes made his famous dichotomy in the 17th century between extension—matter which can be measured—and thought, which cannot be measured, modern man was given a methodology by means of which he thereupon devoted himself, with great emphasis, to the measuring and control of physical nature. The sciences which best fit this method made the greatest strides: physics, for example; later, chemistry; and so on. Thus, medicine made greater strides than psychology and, in the psychological field itself, psychophysics was the most advanced until the last few years. The Newtonian classical physical view that went along with this development is well known. I wish only to emphasize that the philosophical concomitants during this period were, understandably, a great emphasis on rationalism and, later, a devotion of philosophy to an elucidation of scientific method.

During the last part of the 19th century, radical changes occurred in the social systems of Western man. These changes are portrayed in the work of Freud, Marx, Nietzsche, and others, who pointed out that the inherited ways of understanding experience no longer had the same validity. The disunity that became evident during that period in the various sciences is graphically presented by Ernst Cassirer in his lectures at Yale, published under the title *An Essay on Man.* These basic changes and evidences of disunity in modern society had a great deal to do with the emergence of

*This paper was presented at a meeting of the Section on January 17, 1955.

psychology as a centrally important science in its own right.

As is well known, psychology in this country, when it broke off from theology and philosophy in the last half of the 19th century, devoted itself largely to laboratory and experimental work. Though it was kept broad by such leaders as William James, nonetheless, psychology, predominantly in America, has followed until recently the emphasis upon experimental laboratory methods.

The importance of Freud and the later development of clinical and therapeutic psychology can be seen when we realize that Freud sought to find some bases for psychological unity amid the progressively disunified tendencies characterizing persons of the Victorian culture. In essence, Freud pointed out that the rationalistic view of man—that is, man trying to live his life solely by reason and repressing the irrational, unpredictable, and unmeasureable tendencies—was not only unsatisfactory but led to the breakdown of personality.

There is no doubt that clinical psychology and the more intensive forms of psychotherapy are here to stay in our civilization chiefly because of the great and pressing social need for these psychological services.

The bringing together of clinical and traditional scientific psychology presents a number of problems, some of which remain unsolved. I believe that the development of new theory is necessary. This theory in psychology may well be parallel to the new theory in physics as elaborated by Heisenberg, Einstein, Bohr, and others. The essence of the new physical theory is that objective nature can no longer be understood as detached from man. The human being is actor as well as spectator. The parallel to this in psychology would be that we must view man as subject as well as object. I see the new development in physics as symptomatic of far-reaching and necessary changes in our attitude toward man and nature in the Western World. The changes in psychology will occur not because physics now gives us "permission," but rather because of changes running through the basic concepts of Western society.

Psychology needs to work out the theory and method which will be most fruitful for its particular subject matter and aims. The new theory for psychology, which will also serve as a basis for psychotherapy, will include at least the following five aspects:

First, it will preserve the careful methods of experimental psychology. But it will put these methods in a broader understanding of the nature of man. The experimentalist will be aware that, whenever we experiment on a person, we are necessarily leaving out some elements of the whole autonomous human being.

Second, the new theory will include human self-awareness. For we can never see man whole except as we see him, including ourselves, as the mammal who has a distinctive capacity for awareness of himself and his world. Herein lie the roots of man's capacity to reason and deal in symbols and abstract meanings, and herein lies also the basis for a sound

view of human freedom.

Third, the new theory will include some concept of the dynamism of the self. It is not satisfactory to assume the person "grows" like a plant or that the self merely is characterized by a "thrust towards health" like the body. We need some understanding of the aspect of the human being which Allport sought to describe in his term "functional autonomy" and which Goldstein characterizes as "self-actualization."

Fourth, it will include the aspect of culture. For man always lives, moves, and has his being in a social, interpersonal world.

Fifth, the new theory will include the historical dimension of the human being. For man is the mammal who, unlike other animals, can have self-awareness of his past, can transcend the present moment of time, can project himself into the future, thus can learn from the past and, to some extent, can mold his future. It is not generally realized that scientific psychology has treated man almost entirely as an ahistorical unit and, to my mind, this has meant a considerable truncation of understanding.

The sixth aspect of the new theory should be the relation of psychology to ethics. For it is clear from the implications above that man's behavior always influences his own future and impinges upon the rest of the community.

SECTION OF PSYCHOLOGY

THE NEUTRAL THEORY OF PERSONALITY: NEW EVIDENCE AND A REVIEW OF THE ARGUMENT*

By Thomas N. Jenkins

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Since the publication of the neutral theory of personality in 1950,[15] many stimulating and helpful criticisms of the theory have been offered by graduate students, colleagues, physicians, and psychologists. The present report will contain a restatement of the theory, a discussion of some misconceptions of the theory, and a brief exposition of the importance of the theory for diagnosis, prognosis, training, therapy, and test validation, especially in connection with work in military, clinical, and industrial situations.

Critical Exposition of the Theory

THEOREM I. *Primary personality (nonability) traits contain communal variances which comprise two statistically independent basic factors or superfactors.* These superfactors (a term used by Lovell), in Thurstone's[25] technical language, would be referred to as second-order factors. Our investigations, to date, indicate that "these superfactors are neither antagonistic, oppositional, reciprocal, nor bipolar...they are statistically independent behavior tendencies, and hence *neutral* to each other."

The two basic traits described in this report have been labelled *A* and *C*. These labels are the outcome of an early observation that features of personality closely associated with these two superfactors are similar to certain features of personality which some neurologists and physiologists[8, 9, 12, 13, 27] have identified with *adrenergic* (sympathetic or sympathetico-adrenal) and *cholinergic* (parasympathetic or vago-insulin) systems of neural activity. The neutral theory, however, is strictly behavioral in origin. The two superfactors, operationally, are the outcome of factor analyses of the correlations between measures of test response factors. For this reason, these superfactors will be referred to hereafter in this report as *A* and *C* instead of neurologically meaningful terms, such as adrenergic or cholinergic, adrenergy or cholinergy. It will later be shown, however, in discussing the physical basis of the superfactors, that *cholinergic* and *adrenergic* may be both convenient and appropriate adjectives for use in connection with these two basic behavioral tendencies.

*This paper was presented at the meeting of the Section on November 15, 1954.

The close association between certain features of personality and the two superfactors is shown in FIGURE 1. This chart shows a "simple structure" representation of the relationships between superfactors and test scores for a sample of 500 military cadets. It will be noted that factor *I* (superfactor *C*) is clearly associated with frankness, buoyancy (tendency to be elated), emotional spontaneity, arbitrative initiative, organizing initiative, dynamism (tendency to feel lively or energetic), rhathymia, and cautiousness (prudently attentive to dangers or risks). Factor *II* (superfactor *A*), on the other hand, is closely associated with such features as autisms, anxiety, depression, lethargy, hypersensitiveness, shyness, phobias, and inferiority feelings. Note that the factorial axes pass approximately through the centroids of the two groups of test points, and that the factorial axes are perpendicular to each other. This means that the two superfactors common to the two sets of test scores are uncorrelated and, hence, statistically independent of each other. This conclusion is reinforced by the fact that all other large samples investigated to date give similar results. The illustrative sample, discussed at a meeting of this Section in 1950, was a mixed one, comprising 300 men and 200 women, all undergraduate Arts and Science students at New York University.[15]

THEOREM II. *Stable personality trait factors are unipolar, that is, they fail to satisfy the criteria for bipolar simple structure.* This theorem is related to, but not necessary for, the validity of the first theorem. It should be emphasized here that Theorem II refers to traits only; it does not include attitudes. We here follow the lead of Jung[16] and Allport,[2] who clearly differentiated traits from attitudes. Jung, however, refers to basic psychological traits as "psychological functions" (Jung,[16] p. 547). Our analyses have failed to disclose any bipolar trait factors. When test items for antonymous personality features, such as depression and elatedness, were factor analyzed, two outstanding primary factors, instead of a bipolar factor, were revealed. It is worthy of note that some test constructors have treated buoyancy and depression as if they were functions of a single trait continuum, by attempting to measure buoyancy-depression by means of a single scoring category. In fact, both the layman and the psychologist exhibit a strong tendency to describe personality in bipolar terms, a circumstance which is well exemplified in Cattell's classification of lay trait names, and his exhaustive summary of published results based on behavior ratings and self-inventories.[6]

Attention should also be directed to an important corollary to Theorem II, namely, that *factorial tests which seem to be semantically opposite may measure traits which are factorially independent,* that is, they may be uncorrelated The terms buoyancy (tendency to be elated) and depression, for instance, are antonyms, yet the test scores seem to be heavily loaded with superfactors which are functionally independent.

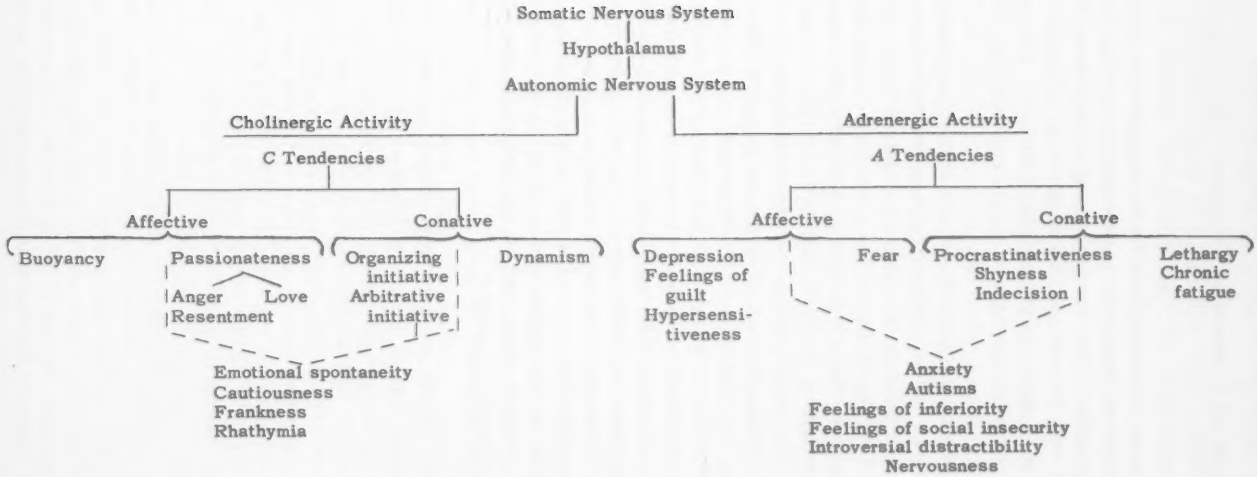

FIGURE 1. A graphical representation of relationships between test scores and superfactors. Numbers within the gaps of the heavy lines are the factor loadings (correlations with the factor).

FIGURE 1, based on a sample of 500 cadets, shows that buoyancy is significantly loaded with superfactor *C* while depression is heavily loaded with superfactor *A*. The correlations between buoyancy and depression scores, moreover, tend to be negligible or low. Thus, antonymous trait names may refer to features of personality which are *not negatively* related, but are functionally independent. If buoyancy and depression were truly antagonistic, opposite, or reciprocal in function, there should be a fairly large negative correlation between them. If completely bipolar, the true negative correlation should be unity.

If we assumed a single (bipolar) trait axis for buoyancy and depression, we might construct a test designed to measure values on such a continuum in terms of negative and positive scores. The zero of the scale, however, could be moved arbitrarily to one end of the distribution of scores. In this case, the scale might be labeled by the test constructor as a test of depression, for which high scores would indicate depressed tendencies and low scores, buoyancy, or elative tendencies. The results of the present investigation, however, strongly suggest that such tests are misleading and fallacious. Since buoyancy and depression are primary factors heavily loaded with independent second-order factors, valid test descriptions of personality necessitate two scoring categories, instead of a mixed category for both buoyancy and depression. As stated in my previous paper on this topic,[15] "a test scale containing both depression and buoyancy items would result in a rather meaningless or ambiguous composite score for the two traits. This possibility may account for some of the difficulties in using current personality tests effectively in clinical work, and in the selection and allocation of industrial and military personnel."

(1) *Difficulties in the interpretation of test patterns.* The semantic paradox obviously implies certain difficulties of interpretation of the neutral theory of personality when evaluating individual patterns of test scores. Let us suppose that an individual obtains high scores for both buoyancy and depression. Now let us examine three possible interpretations of these test scores, as follows: (1) the individual may be extremely happy and sad at the same time; (2) he may be, for no apparent reason, very happy at certain times and very sad at other times; and (3) he may be highly reactive to happiness-producing situations and may be also highly reactive to depressing situations. The first suggested interpretation is unsatisfactory, but is mentioned here because students sometimes think that this interpretation is what the neutral theory means. The second suggestion is a more plausible one. If an individual exhibits periodic swings between happiness and unhappiness, we have an example of what is sometimes referred to as cycloidism. Clinical and analytical data, however, indicate that the third interpretation is the most tenable

one for healthy persons. A well-adjusted, realistic individual may be highly reactive to both happy and depressing situations.

According to our third mode of interpretation, various other patterns of buoyancy and depression could be explained as follows: (1) a high buoyancy score and low depression score would indicate high reactivity to happy situations, and nonreactivity or "no affect" with respect to depressing ones. Such an individual would appear to the untrained observer as a chronically happy person. (2) A person receiving low scores on both scales would be one whom clinicians sometimes refer to as an individual "without affect." (3) An individual may obtain a validly high score for depression, and a low one for buoyancy. Such an individual would be highly reactive to depressing situations, would react very little to happy ones, and would be likely to be erroneously characterized by certain observers as a "depressed personality type."

(2) *What is a bipolar factor?* Doctor Raymond B. Cattell has kindly reminded me of a source of confusion in this connection. The confusion seems to arise because of the difference between Burt's [4] residual bipolarity concept and Thurstone's [25] concept of bipolar simple structure. Centroid methods of factoring are averaging procedures. The deviations or residuals around an average are either positive or negative. When the first factor is removed from a correlation table by means of an averaging procedure, what remains of the table of correlations comprises a set of negative and positive deviations. Hence, after removal of the first factor, succeeding factor loadings may be either positive or negative. Burt refers to factors computed after removal of the first factor as bipolar, and prefers to render a bipolar interpretation of such factors.

Thurstone, on the other hand, tends to get rid of negative loadings by rotation of the factorial frame of reference. This tendency is well exemplified in Thurstone's [26] factor analysis of the 13 scores of the Guilford-Martin series to determine the number of dimensions in these personality schedules. "We started the analysis," he writes, "with the expectation of finding bipolar factors for all or most of these factors; but the result revealed all of them to be positive."

The following principles may be stated for defining a bipolar factor in terms of an underlying function or psychological structure: (1) hypothesize that certain paired features of personality, such as buoyancy and depression, are bipolar, and prepare measures for the paired features; (2) intercorrelate these measures; (3) after factoring, attempt to rotate the factor axes so as to define the positions of the test points in positive terms without arbitrarily changing the signs of the test scores by reversing (reflecting) the test vectors; and (4) adjust the positions of the factor axes to reduce the absolute sum of the products of the paired test loadings (coordinates of each test point) to a minimum. If a real bipolar psychological function is involved, then bipolar simple structure

should be revealed. *In other words, a real bipolar psychological function cannot be rotated out of existence.* The third set of factor loadings shown in TABLE 1 may be such a factor. It is a small component, comprising less than 9 per cent of the total common factor variance. If tests 7, 8, 19, and 22 were eliminated, factor III would constitute only 4.3 per cent of the total factor variance. It probably is attitudinal in nature, and possibly may be a common factor of the *invalid* component of the test scores. Further research, preferably of a hypothetico-deductive nature, would be required to determine the meaning of the third factor.

(3) *Interpretation of the superfactors.* The meaningful significance of *A* and *C* for theory and practice will depend on additional research. Probably the most effective in this regard would be a combination of clinical, laboratory, and psychometric approaches in dealing with criterion populations. Eysenck[11] states that the traits closely associated with the *C* superfactor are "a group of typically extravertive traits," and suggests

TABLE 1

UNROTATED FACTOR MATRIX FOR 25 TEST SCORES
(The Rotated Factor Loadings are Shown in FIGURE 1)

	Test	I	II	III	h^2
(1)	Frankness	.15	-.37	.17	.19
(2)	Buoyancy	.25	-.49	-.26	.37
(3)	Emotional spontaneity	.31	-.60	.12	.47
(4)	Arbitrative initiative	.18	-.56	-.13	.36
(5)	Organizing initiative	.26	-.55	-.20	.41
(6)	Dynamism	.14	-.65	.04	.44
(7)	Rhathymia	.21	-.29	-.45	.33
(8)	Cautiousness	.09	-.34	.39	.28
(9)	Obsessive autisms	.74	.20	.04	.59
(10)	Depression	.56	.31	.12	.42
(11)	General anxiety	.74	.31	.13	.66
(12)	Social insecurity	.69	.22	.14	.54
(13)	Hypersensitiveness	.71	.32	.15	.63
(14)	Self-commiserative autisms	.76	.24	.14	.66
(15)	Lethargy	.47	.33	-.15	.35
(16)	Nervousness	.64	.29	.16	.52
(17)	General inferiority	.62	.34	.11	.51
(18)	Face-to-face shyness	.49	.29	.18	.36
(19)	Procrastinativeness	.41	.32	-.30	.36
(20)	Introversial distractibility	.62	.33	-.16	.52
(21)	Feelings of guilt	.65	.19	.10	.47
(22)	Specific anxiety	.66	.27	.32	.61
(23)	Unpleasant reminiscence	.66	.13	.09	.46
(24)	Phobias	.59	.14	.04	.37
(25)	Color shock	.53	.06	.13	.30
	Total factor variances	7.04	3.15	.96	
	Per cent of grand total	63.1	28.3	8.6	
	Total var. without tests 7, 8, 19, & 22	6.38	2.78	0.42	
	Per cent without tests 7, 8, 19, & 22	66.7	29.0	4.3	

that the *A* superfactor probably can be identified as Eysenck's neurotic dimension.

There are cogent reasons, however, for grave skepticism concerning Eysenck's interpretation of the superfactors. Various writers, such as Allport, Eysenck, Maurer, and MacDougall, have referred to many of the traits associated with superfactor *A* as indicators of introversion Among these are the autisms or fantasies, shyness, hypersensitiveness, feelings of inferiority, anxiety, and depression. If superfactor *C* were interpreted as extraversion, then the so-called introversial *A* traits should be closely associated with *C* on *opposite* poles of a bipolar continuum instead of being identified with a neurotic dimension. It would be much safer to assume that neither *A* nor *C* refer to the attitudinal dimension of extraversion-introversion. In this case, one might be justified in interpreting superfactor *A* as a neurotic dimension, or possibly a depression, anxiety, psychasthenic, or dysthymic dimension. It should be emphasized again, however, that verified meanings other than syndromal descriptions depend upon additional research Later in this report, a physical interpretation of *A* and *C* will be suggested.

Helpful clues concerning the possible existence of an attitudinal superfactor are furnished by the loadings for small factor *III* in TABLE 1. Limiting our discussion to third factor loadings of .30 or greater, we find that rhathymia and procrastination are negative, while cautiousness and specific anxiety are positive. Thus, it will be noted that the *A* and *C* loadings are not related in a systematic manner to the negative and positive loadings of the third factor, a fact which is clearly revealed in the following table of rotated factor loadings:

	I (*C*)	*II* (*A*)	*III*
Cautiousness	.35	-.05	.39
Specific Anxiety	.01	.71	.32
Rhathymia	.35	.08	-.45
Procrastinativeness	-.13	.51	-.30

If factor *III* be interpreted as an extraversive-introversive dimension, then we can conclude that *A* or *C* potentials each may be handled either way, that is, in an extraversial or an introversial manner. For example, let us assume that negative third-factor loadings (rhathymia and procrastinativeness) are extraversial, and that positive third-factor loadings (cautiousness and specific anxiety) are introversial. Then cautiousness, a *C* trait, and specific anxiety, an *A* trait, would both be introversial.

Nature of the Behavioral Evidence

As indicated previously,[15] the present theory is the outcome of an extensive exploratory investigation of personality, initiated in 1942, involving both laboratory and test studies. The data presented in this

report, however, are based primarily on human behavior in test situations. Retholtz,[20] on the other hand, working in the New York University laboratories, obtained results for changes and individual levels of skin resistance, blood pressure, sway, suggestibility, tremor, and Schneider's index, which lend support to a neurological interpretation of the *A* and *C* superfactors.

Various types of self-appraisal response scales were tried out on large experimental populations. These comprised broad and narrow response scales, and appraisals in terms of trait loading (how much?), frequency (how often?), and attitudes. Broad response scales were finally used for two basic reasons: (1) there is less feeling of response restriction on the part of testees; and (2) the broad scale furnishes the basis for a more advantageous cut for statistical treatment or scoring. The frequency and trait loading scales gave results which correlated highly with each other, but the frequency response scale proved to be the more satisfactory for wide ranges of intelligence. Testees, in other words, find it easier to make a judgment in terms of frequency. The face validity of the items varied from high to little or no face validity. An example of a low face validity item would be the following: "I enjoy the sound of dripping water."

The data in this report are based on a factorial test battery of 720 items.* Responses were made to a six-point, closed-frequency scale, ranging from *always* to *never*. The closed type of scale tends to minimize the effect of one type of variance, namely, subjective notions concerning the width of the scale. The scale consisted of letters which stand for the following: *always, almost always, often, sometimes, hardly ever,* and *never*. The form of the item and scale is shown in the following example:

A AA O S HE N 109. Eating between meals makes me feel calm.

TABLE 1 is the result of the complete centroid method of factoring. To minimize the influence of test attitudes on the results, test reticence, based on a set of 53 items, was partialed out of the intercorrelations of the other test scores. The resulting table of partial correlations was then factorized. FIGURE 1 is a graphic representation of superfactors *I* and *II*, after rotation to a meaningful configuration of simple structure. The numbers at the broken portions of the heavy lines are the loadings, that is, the correlations with the factor. It will be noted that correlations with superfactor *I* (*C*) are 50 or better for the traits of buoyancy, emotional spontaneity, arbitrative and organizing initiative, and dynamism. The largest factor correlations, however, occur in connection with superfactor *II* (*A*). Factor correlations of .75 or better, for instance, are revealed for the autisms, general anxiety, and hypersensitiveness Similar results have been obtained for samples drawn from diverse populations and for

*At present, parts of this battery are being revised; the complete battery will be ready for publication late in 1955.

both men and women. As stated heretofore, the sample discussed in the 1950 report was a mixed group, consisting of 300 young men and 200 young women [15]

Some Applications of the Neutral Theory

(1) Environmental versus constitutional factors. It is likely that significant factors of the second-order or higher are more closely related to the constitutional make-up of the individual than those components which differentiate personality potentials into primary factors. The primary factors which have been analyzed in this report actually contain first-order and second-order (general) components. If these two sets of components were separated and measured, it would probably facilitate the attempt to differentiate accurately between the genotypic (constitutional) and phenotypic (directly observable) aspects of human behavior in many psychological domains. If the superfactors in our study were separated from the primary factors, the remainder would constitute a set of independent dimensions, which Cyril Burt[4] refers to as *group factors*. As examples, let us consider the true standard primary scores for depression, hypersensitiveness, and lethargy. If the true superfactor scores for the individual be subtracted from these primary scores, the resulting values (group factor scores) would be the components which truly differentiate depression, hypersensitiveness, and lethargy from each other. These values, moreover, would be unrelated to each other, that is, they would be statistically independent. It is recommended, therefore, that separate scores be developed for *group factors* and *superfactors*, instead of having the two sets of effects mixed together in the form of primary scores. In a given individual, a superfactor may be small and some *group* factor scores be large, while the reverse may be true in other individuals.

(2) Importance for clinical work. The measurement of primary traits in terms of group factor and superfactor scores should aid the clinician in diagnosis, prognosis, and therapy. If, for instance, the *A* score were large, the best therapeutic approach probably would be different from the case where the *A* score was small and certain *group* factor scores were large. In the former, the usual professional procedures plus conditioning or re-education might be in order while, in the latter case, a radical change of environment might be a first step in therapy. For instance, a low score for superfactor *A*, and a high group factor score for feelings of social insecurity would indicate a different mode of handling the case than if the reverse were true; that is, if the predominantly genotypic factor were small and social insecurity large, then the maladjustive condition of the client might be due to inability to obtain employment, while a large genotypic (superfactor *A*) would suggest a constitutional disposition to feel insecure, inferior, or anxious. The

relative loading of *A* and *C* in the individual also may be very important for diagnosis and prognosis; moreover, individuals heavily loaded with both *A* and *C* are the most likely to be misunderstood by both untrained and professional observers. A man with low *A* and *C* scores, on the other hand, would probably be characterized by a clinician as an individual "without affect."

Cronbach,[7] in comparing clinical work with exact laboratory or psychometric measurements, uses an analogy from Shannon's treatise on communication theory, namely, that bandwidth and fidelity are inversely related to each other. The clinical approach, he argues, is a wide-band approach. In some respects, this is an unfortunate analogy. It is true that the clinician must make over-all appraisals, and he is usually in a position where he has to make decisions. But there is no inherent conflict between the wide-band approach of the clinician and precision in dealing with clinical problems Clinicians undoubtedly would welcome any aid to precision which could be integrated into a wide-band approach. A comprehensive set of measures of primary personality factors, or group factors and superfactors, should materially aid the clinician in fortifying his judgment.

(3) Superordinate and subordinate behavior. Military and industrial psychology is complicated and, in some ways, is fraught with as many difficulties as clinical psychology. Both of these disciplines should be mature enough to begin determining the potentials for success in terms of well-defined personality factors. In the military and industrial domains, both managerial and subordinate behavior are very important. TABLE 2 shows that individuals with initiative tend to be heavily loaded with superfactor *C*. But it would be a mistake to assume that superfactor *C* or a few leadership qualities will make a successful leader. For instance, note that domineeringness is also heavily loaded with *C*, but in most situations such behavior would be contraindicated in a democratic society.

(4) The authoritarian personality. Adorno *et al.* [1] have attempted to depict the essential features of a personality type which they call the authoritarian personality. According to this view, the authoritarian exhibits a submissive, uncritical attitude toward idealized moral authorities of the in-group, but shows a domineering, hypercritical tendency to condemn, reject, or punish those who violate conventional values. Returning to TABLE 2 (set IV), we note that it is a little difficult to harmonize this point of view with the *A* and *C* loadings of the traits in the authoritarian syndrome Domineeringness is heavily loaded with *C*, while hypercriticalness is significantly loaded with both *A* and *C*. Submissiveness, on the other hand, is loaded primarily with superfactor *A* If the authoritarian is high on both domineeringness and submissiveness, he, therefore, must be heavily loaded with *both* superfactors. An important key to his personality organization may be cathectic obedience.

TABLE 2

SHOWING SIMPLE STRUCTURE LOADINGS OF SUPERFACTORS *A* and *C* for a Select Set of Variables

	Test	*C*	*A*
I	Seriousness	.31	.48
	Cautiousness	.35	.05
II	General inferiority	.07	.70
	Social insecurity	.06	.72
	Superiority feelings	.46	.27
	Grandiose autisms	.24	.68
III	Arbitrative initiative	.59	.05
	Organizing initiative	.61	.03
IV	Domineeringness	.62	.10
	Hypercriticalness	.36	.48
	Submissiveness	.06	.41
	Cathectic obedience	.26	.33
V	Color shock	.15	.51

Liking to obey (positive hedonic cathexis) is an attitudinal score, but it seems to be significantly loaded with both superfactors, a positive loading for *C* and a negative loading for *A*.

(5) Geriatrics and longevity. Dunbar,[10] in her attempt to find a very healthy sample of people, made an intensive study of the life history, habits, and personality of very elderly men and women. Some of her descriptions of centenarians suggest that these individuals are much more heavily loaded with superfactor *C* than with superfactor *A*. If such be the case, it is likely that this fact is closely associated with the constitutional make-up of these individuals Stern's[14] discussion of reactive depressions in later life suggests that knowledge of the *A* and *C* loadings in persons may be helpful in determining how they will react to and handle the frustrations of old age.

(6) Interpretation of projective test results. The neutral theory can be helpful in ascertaining the deeper significance of some of the widely publicized features of projective tests, as, for example, the Rorschach. To illustrate this point, color shock was included as a part of the present report. TABLE 2 shows that the correlation of color shock with superfactor *C* is only .15, while with superfactor *A* it is 51. This difference is graphically depicted in FIGURE 1. Studies of this sort could be readily extended to other features of projective tests. It would be one way of quickly revealing some of the basic meanings of projective test results.

(7) Are superiority and inferiority feelings bipolar? The terms "superiority" and "inferiority" are opposite in meaning. Analytic and psychoanalytic schools of psychopathology might assume that superiority feelings are indicators of an unconscious sense of inferiority. Inspection of TABLE 2 (set II), on the contrary, reveals the fact that superiority

and inferiority feelings are functions of two independent dimensions, namely, superfactors *A* and *C*. It will be noted that feelings of inferiority and social insecurity are very heavily loaded with *A*, while superiority feeling is a function of both *A* and *C*, but is more closely related to *C* than *A*. Grandiose autisms, on the other hand, are more closely related to superfactor *A*.

(8) Factorial definitions of personality variables as an aid to neurophysiologists. Factorial measures should enable neurologists to associate anatomical and physiological observations more closely with quantitatively determined behavioral response factors or superfactors. Moreover, it probably would facilitate greater agreement regarding behavioral terminology among neurologists, psychiatrists, clinical psychologists, and experimental psychologists.[18] A clinician,[13,24] for instance, may consider anxiety as fearful anticipation or apprehensiveness, while a neurophysiologist may implicitly define anxiety as angry or aggressive anticipation of something unfavorable Wolf and Wolff,[27] for example, imply the latter meaning by assuming that anxiety is closely related to feelings of resentment and hostility. Measures of psychological factors would enable the neurophysiologist to relate neural structure and physiological function to quantitatively isolated and measured traits, especially second order or higher, such as superfactors *A* and *C*.

(9) Nonfactorial tests and expert opinions. The factorial approach helps to define properly those features of behavior which, to the practical observer, *seem* to reflect the same basic underlying traits. Cautiousness and seriousness are likely to be classified together, yet TABLE 2 (set I) shows that they differ considerably even from the standpoint of second-order factors. Cautiousness is not related to superfactor *A* while seriousness is in the *A C* plane, and appears to be more heavily loaded with *A* than *C*.

Some tests are carefully developed by selecting items which agree or correlate with expert opinion. When expert opinion is utilized in this way, the test-maker refers to it as an external criterion. Outstanding in this respect is the Minnesota Multiphasic Personality Inventory, where the expert opinions were furnished by psychiatrists. Such a test predicts what psychiatric opinion is most likely to be.

Test items also may be selected exclusively on the basis of expert opinion. Expert selection of items may also serve as the validating criterion An example of this type is the Taylor test of anxiety.[24] A group of clinicians selected those items from the MMPI, which they thought were indicators of anxiety. Then 65 items were selected for which the experts showed 80 per cent agreement or better. This test, without further validation, was then used in an eye-lid conditioning experiment to determine the relationship between conditioning rates and levels of anxiety. Since then, Bitterman, Holtzman, and others have failed to find a significant relationship between Taylor scores and

clinical ratings of anxiety.[3] Sampson and Bindra[21] recently studied 51 male, hospitalized neurotics, 27 of whom were diagnosed as "anxiety neurotics" and 24 as "non-anxiety neurotics." They claim that scores within a limited range (19-33) are likely to be associated with a diagnosis of anxiety, while extremely high scores are associated "mainly with depression."

This observation of Sampson and Bindra suggests the kind of validative errors which may exist in a test based on expert opinion and no factorial validation. A person may be depressed without being anxious. *Grief* and *dread*, moreover, are not synonymous. Alexander[14] has emphasized the importance of clearly distinguishing between depression and anxiety, when deciding whether to use convulsive electroshock treatment. Inspection of the Taylor test for content reveals such behavioral categories as the following: alimentary disorders, nervousness, worries, sweating, fears, hypersensitiveness, depression, buoyancy, feelings of inferiority, lack of confidence, procrastination, indecision, irritability, weeping, and lethargy. It will be noted that these qualities tend to correspond to the *A* traits listed in FIGURE 1. By lumping these categories together, the Taylor test fails to give a measure of anxiety. It gives instead a rather *crude measure of superfactor A*. Much of the confusion that has ensued since the publication of the Taylor test could have been eliminated at an early date through careful factorial validation. This example serves to emphasize the importance of factorial validation. In fact, in developing personality scales, it would be much safer to utilize both factorial and external validation and, for the sake of economy of effort, undertake factorial before external validation.

Similar comments might be offered for behavior ratings of manifest anxiety. Kendall,[17] for instance, made use of nine so-called subtraits descriptive of manifest anxiety drawn from Cameron's[5] description of chronic anxiety. These nine aspects (fatigue, alimentary disorders, upset, tension, restlessness, *etc.*) apparently were meant to agree substantially with those covered by the Taylor scale. Nurses were asked to rate patients on the basis of the "nine aspects of manifest anxiety." It is not clear just how the ratings were combined, but here, again, a combination of such ratings does not result in a true evaluation of anxiety but, instead, in an indirect and very crude evaluation of superfactor *A*.

(10) Affective and conative relationships with the two superfactors. It is interesting to note that both emotional and motivational traits are related to these basic behavior tendencies. The facts in support of the neutral theory, however, tend to show that emotional and motivational tendencies may or may not be related to each other. Inspection of FIGURE 1 reveals the fact that affective traits, such as buoyancy, and conative traits, such as dynamism and organizing initiative, are

related through a common basic factor, namely *C*. Emotional traits, such as depression and fear, and motivational traits, such as lethargy and procrastinativeness, are closely related to *A*. If we were using neurochemical terms, we might divide affective-conative tendencies into two large categories, the cholinergic and adrenergic. The neutral theory tends to throw the interrelationships of affective and conative traits into proper perspective.

Physical Basis of the A and C Superfactors

Recent studies[12-14, 27] suggest that chronic fatigue, chronic anxiety, fear, and depression are closely associated with the sympathetic component of the autonomic nervous system, while happiness, optimism, resentment, feeling of well-being, and dynamism tend to be associated with the parasympathetic component. As pointed out elsewhere,[15] considerable confusion seems to have arisen because of a rigid adherence to anatomical origins "Darrow[9] was one of the first investigators to show that behavior which is parasympathetic in character may be mediated through postganglionic sympathetic fibers. He noted that stimuli producing marked changes in palmar resistance induced very little change in blood pressure, while situations eliciting large changes in blood pressure resulted in very little change in palmar resistance. The sweat glands are innervated by sympathetic nerves, but activated through the release of acetylcholine. Acetylcholine, however, is usually released by postganglionic parasympathetic fibers, while adreninlike substances are usually, but not always, produced by the postganglionic sympathetic fibers. Dale,[8] in 1933, suggested that activation of effectors by means of acetylcholine should be called cholinergic, regardless of the anatomical origin of the neural conductor. He suggested the term 'adrenergic' for activation by adreninlike chemicals such as sympathin."[15]

If the *C* superfactor is closely associated with cholinergic or parasympathetic activity, and the *A* superfactor with adrenergic or sympathetic activity, it would be very convenient to refer to these factors, respectively, as *cholinergic* and *adrenergic*. The two sets of behavior tendencies could be referred to as cholinergy and adrenergy If this hypothesis be true, then a person with high *C* trait scores and low *A* trait scores would exhibit the behavioral signs of cholinergic dominance. Both human and other animal experiments indicate the possibility of parasympathetic or sympathetic dominance. Cholinergic individuals would probably react very differently from adrenergic ones to stressful situations. Gellhorn,[12] for instance, in referring to Richter's observation that during the London air raids, some faces turned white while others flushed, "suggests a sympathetic dominance in the first and a parasympathetic dominance in the second group"

A suggested schema of relationships between the *A* and *C* superfactors

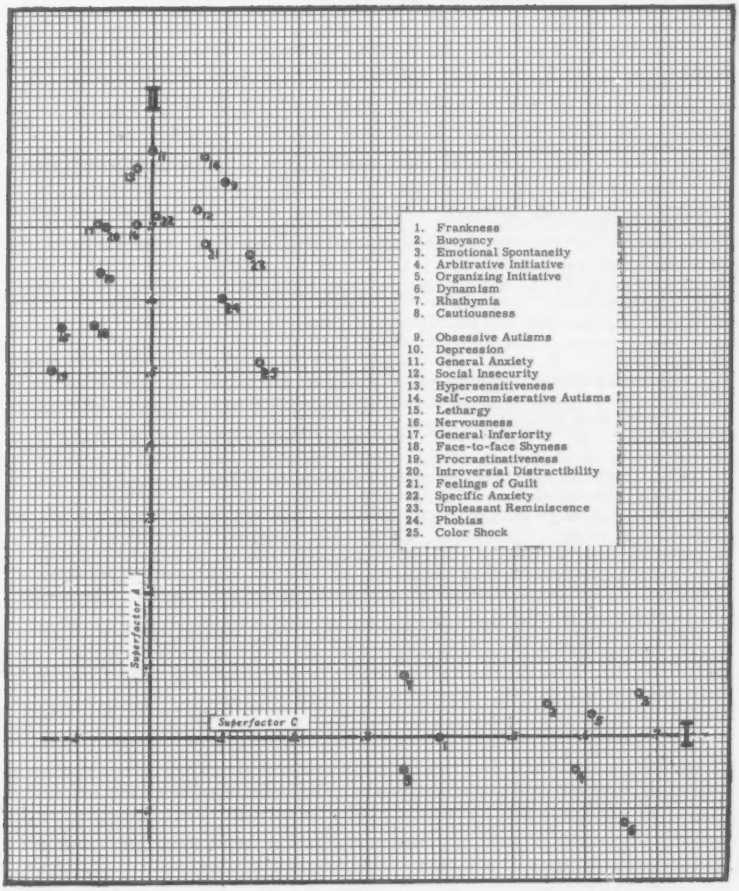

FIGURE 2. Schema showing suggested neural relationships between affective or conative traits, and superfactors *A* and *C*.

and the nervous system is shown in FIGURE 2 This schema also suggests how both affective and conative aspects of behavior may be related to the autonomic nervous system.

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SECTION OF MATHEMATICS AND ENGINEERING

MODERN MATHEMATICS FOR SCIENTISTS AND ENGINEERS: BOOLEAN ALGEBRA*

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Before discussing Boolean algebra, I shall have to say something about the enormous change in *attitude* which took place in mathematics around 1800, for this change in attitude was basically responsible, not only for the development of Boolean algebra, but also for many other contributions to modern mathematics—an overwhelming flood of them. Indeed, as is pointed out by E. T. Bell, the distinguished American mathematician, if the *History of Mathematics*, by M. Cantor, which goes up to the year 1800 and is in four volumes, were continued to cover the 19th century in comparable detail, this additional material would require 19 volumes, almost 5 times as many as were required by Cantor to cover the mathematics of all previous centuries. And this history would not include the mathematics of the 20th century, which is increasing at an ever greater rate.

Now what was the change in attitude that led to this enormous increase in mathematical productivity? In order to understand this change clearly, let us go back for a moment to the work of Euclid, with which we are all familiar. As every schoolboy knows, Euclid, in about 300 B. C., gathered together the then-known geometrical knowledge, consisting of isolated theorems such as the Pythagorean theorem and others into a *system* that has been the model for all scientific systems since then. And what is the main characteristic of such a system? We all know that it is the separating off of some of the theorems as "postulates" or "axioms," from which, by means of logic, all the remaining theorems may be derived. Now since the "proof" of a theorem means its derivation from previous theorems, the first group of theorems (or postulates), therefore, cannot be proved, since there is nothing preceding them from which to derive them. Thus, any system *must* start with *unproved* theorems and, by the same token, also with *undefined* terms, from which all the other theorems will be proved and all subsequent terms defined. This observation always comes as a shock to the layman who, though he is unacquainted with mathematics, still worships it at a distance, believing that *there*, at least, is a domain in which one can prove everything, and which is therefore absolutely reliable. Euclid himself, however, understood very well that his system rested on unproved propositions and undefined terms. Why, then, did he believe in the reliability of his system? What was his idea of the *nature* of the postulates themselves? How could they serve as a start-

*This paper was presented at a meeting of the Section on January 21, 1955.

ing point if they themselves could not be proved? The answer for him was, of course, that the postulates were "self-evident truths." In other words, any proposition is either (1) a "self-evident truth" or (2) something which must be proved, *i.e.*, derived from "self-evident truths" or from propositions which have already been derived from such truths, as shown diagrammatically in FIGURE 1.

Among the propositions which Euclid listed as postulates was the well-known "parallel postulate" which may be stated thus: "Through any given point which is not on a given line, one, and only one, line can be drawn which is parallel to the given line." Though Euclid listed this proposition among his postulates, he did so reluctantly, because it did not seem to him to be so "self-evident." Hence, he set about to prove it from his other postulates, but without success. Other mathematicians then made further attempts to prove it but, over a period of many centuries, all these attempts met with failure. In the early 19th century, after 2,000 years of failure, it dawned on several mathematicians at about the same time, that the reason for this failure was a wrong *attitude* toward the *nature* of postulates. That is to say, mathematicians realized that postulates should not be regarded as "self-evident truths" at all, but rather as mere man-made assumptions. Accordingly, the diagram in FIGURE 1 should be replaced by the diagram shown in FIGURE 2.

Now does it really matter what the philosophical attitude is toward the nature of postulates? Let us apply this question of attitude to the problem of the parallel postulate. Since the parallel postulate is not proved, and is to be regarded not as a "self-evident truth" from which there is no escape, but rather as a mere man-made assumption, we might discard it altogether and replace it by an entirely different assumption, namely: "Through any given point which is not on a given line, *two* lines can be drawn which are *both* parallel to the given line." Fantastic? Ridiculous? Not at all—as you will soon see. Note that the very concept of a "man-made assumption" gives us the freedom and the courage to change it and follow through logically to the consequences of the new assumption. This is exactly what was done by the above-mentioned mathematicians at the beginning of the 19th century—namely, by Lobachevsky, Bolyai, and Gauss, all coming to the same conclusions although working independently of one another (as has so often happened in the history of mathematics). And what was the result? They succeeded in developing a *new* kind of geometry, a non-Euclidean geometry in which the parallel postulate was different from Euclid's by assuming two lines, instead of only one, through a given point, parallel to a given line (see above); all the other Euclidean postulates were retained. This led to some new unfamiliar theorems such as "The angle-sum of a triangle is *less* than 180°," *etc.*[1] This very strange geometry was later shown by Beltrami, in 1868, actually to apply on a certain curved surface (a "pseudosphere"[1]) rather than on the more familiar "flat" surface of Euclid's

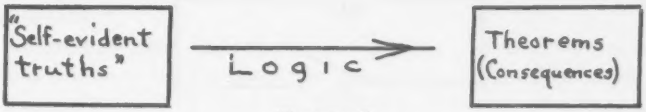

FIGURE 1

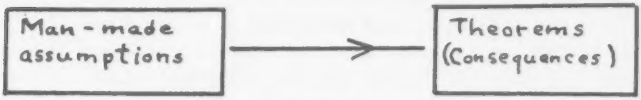

FIGURE 2

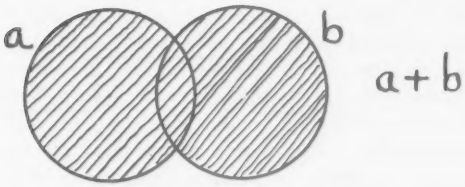

FIGURE 3

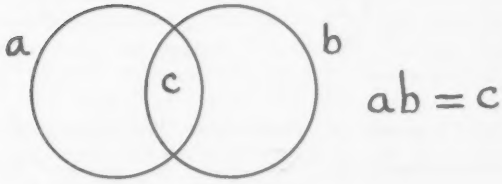

FIGURE 4

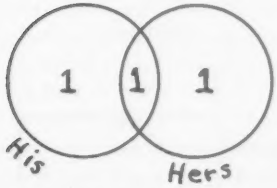

FIGURE 5

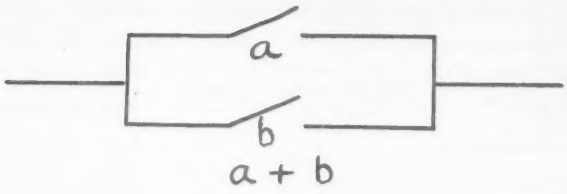

FIGURE 6

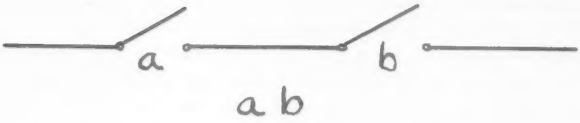

FIGURE 7

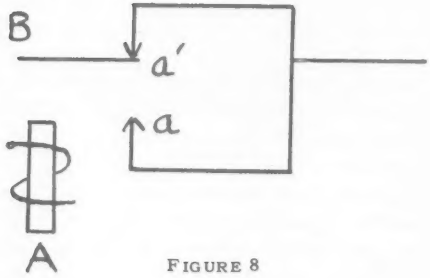

FIGURE 8

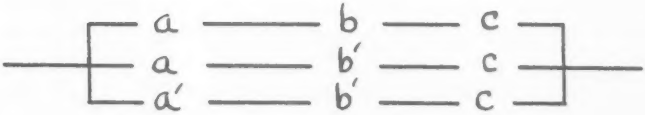

FIGURE 9

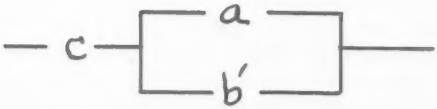

FIGURE 10

geometry. Not only that; it is this non-Euclidean geometry which applies to our physical universe and was used by Einstein in his *General Theory of Relativity*.[2]

Furthermore, about 1850 the famous parallel postulate was again replaced, this time by Riemann, who postulated: "Through a given point not on a given line *no* lines can be drawn parallel to the given line." This development led to another non-Euclidean geometry which was subsequently shown, also by Beltrami, actually to hold on a sphere, on which the angle-sum of a triangle is *more* than 180°, *etc.*[1] Thus, the view that postulates are mere man-made assumptions and are therefore subject to change was fully justified. Indeed, this was only the beginning, for now mathematicians began to examine also the postulates of algebra, to change them, and thus to achieve other new algebras (among them Boolean algebra, about 1850). In fact, a whole flood of new mathematical systems was inaugurated, many of which already have had useful applications, and many more such systems will undoubtedly be applicable in the future.[3]

One use for such systems is to prove the "independence" of a given postulate in a system. Thus, even if the change in Euclid's system had not turned out to be essential in the study of the physical universe (*e.g.*, by Einstein, as already cited), it would still have been useful in that it showed Euclid's parallel postulate to be "independent" of the other Euclidean postulates. In other words, this parallel postulate is *necessarily underivable* from these other postulates, since its contradictories (the one allowing two parallels and the one allowing no parallels at all—see above) can also exist in harmony with those other postulates, as in the non-Euclidean geometries mentioned. No wonder the mathematicians could not prove Euclid's parallel postulate—even though they had the colossal patience to keep trying for 2,000 years. Similarly, Huntington[4] used this idea to prove the independence of the postulates of ordinary algebra, *i.e.*, the algebra of complex numbers. Thus he lists 27 postulates for this algebra, and then proceeds to make up 27 different "algebras," each one differing only with respect to one postulate from his original set, thus proving their independence, as in the story of the parallel postulate told above.

Now that we appreciate the possibility of creating *various* mathematical systems—and this is the *Great Discovery* of the early 19th century from which the other discoveries flowed, including geometries, algebras, arithmetics, *etc.*—let us turn our attention to Boolean algebra in particular, and to some of its important applications.

First of all, in the spirit of "postulational thinking" discussed above, the reader, of course, will want to see a postulate set for Boolean algebra:[5]

(1) "Closure" for addition $a + b = c$, *i.e.*, adding two elements of the system gives a result which is itself an element of the system.

(2) Closure for multiplication: $ab = c$.
(3) $a + 0 = a$
(4) $a \times 1 = a$
(5) $a + b = b + a$
(6) $ab = ba$
(7) $a(b + c) = ab + ac$
(8) $a + (bc) = (a + b)(a + c)$
(9) $a + a' = 1$
(10) $aa' = 0$
(11) There are *at least two* different elements in the system.

Now, if we try these postulates on ordinary algebra, we shall find that some of them work (such as Nos. 1 through 7), but some of them (such as No. 8) do *not* work, and others (such as Nos. 9 and 10) are meaningless. But realizing that all these postulates are man-made assumptions, we know, of course, that we have the *right* to make them and to derive theorems from them, provided only that there are *no contradictions* in the system, for contradiction is the only sin in mathematics—which definitely cuts out lying as a successful tool of thought! Further, if we define the elements (namely a, b, c, 1, 0, a', *etc.*) in certain ways, we find that all these postulates work, and we shall be able to use this algebra for various purposes.

Let us first take the elements to mean "classes" of objects, which we shall represent by circles called "Venn" diagrams. Now the *sum* of two classes, a and b, is defined by the shaded area in FIGURE 3, and the *product* of two classes is that part which they have in common (FIGURE 4).

Here $ab = c$.

Let me illustrate this "algebra of classes," Boolean algebra, with a simple homely example.

Suppose a widower with one child marries a widow who also has one child, and then they beget another child by the new marriage. Now represent diagrammatically (by a "Venn" diagram) the class of his children, the class of her children, and the class of *their* children, as in FIGURE 5.

Here we can easily see that

$$2 + 2 = 3 \text{ (\textit{not 4}, you notice)}$$

and

$$2 \times 2 = 1 \text{ (!)}$$

(Remember the definitions of *sum* and *product* of classes given above.)

What then becomes of the "eternal verity" that 2 and 2 always make 4? We see that, in mathematics, "eternal verity" has been replaced by the more fertile idea of "freedom to adjust" for *survival* and *growth*. But please note that this new-found freedom in mathematics is under strict control and is not just wanton license. An application of Boolean algebra to a very "practical" problem has been made by F. E. Hohn, in the Bell Telephone Laboratories,[6] to electrical circuits in the following manner:

Let a, b, c, *etc.*, represent switches; let 1 represent the condition of a switch being "closed," and 0 the condition of a switch being "open."

Further, let the *sum* of two switches mean connecting them in parallel, as in FIGURE 6, and let the *product ab* mean connecting them in series, as in FIGURE 7.

Since any switch can be only "open" or "closed," we can have only the following possible combinations for a pair of switches:

Sums	*Products*
$0 + 0 = 0$	$0 \times 0 = 0$
$1 + 0 = 1$	$1 \times 0 = 0$
$0 + 1 = 1$	$0 \times 1 = 0$
$1 + 1 = 1$	$1 \times 1 = 1$

which means, of course, that when the switches are connected in parallel (sum), then the entire circuit $(a + b)$ is "open" $(= 0)$ only if *both* switches are "open" $(= 0)$; and when the switches are connected in series (product), then the circuit is "open" $(= 0)$; that is, the current cannot flow, in all cases, *except* when *both* switches are "closed" $(= 1)$. Both tables given above, it should be noticed, are in agreement with the postulates for Boolean algebra (page 335). Finally, a' is defined as shown in FIGURE 8. Here A is an electromagnet which, when activated, attracts the spring B, thus making an electrical contact at a and breaking it at a', and when A is not magnetized, spring B jumps back and makes contact at a' and breaks it at a. If we now have an electrical circuit as shown in FIGURE 9 with nine contacts, we may represent it algebraically (in Boolean algebra) by the function:

$$f = abc + ab'c + a'b'c$$

(See Hohn's definitions of *sum* and *product* cited above and apply them to the diagram in FIGURE 9.)

We may transform this function as follows:

$f = c(ab + ab' + a'b')$	by postulate 7
$= c[a(b + b') + a'b']$	by postulate 7
$= c[a(1) + a'b']$	by postulate 9
$= c(a + a'b')$	by postulate 4
$= c(a + a')(a + b')$	by postulate 8
$= c(1)(a + b')$	by postulate 9
$= c(a + b')$	by postulate 4

which is, of course, equivalent to the given function and will produce the same desired effect. If we translate this back into a diagram, we have the situation shown in FIGURE 10 with only *three* contacts, thus resulting in a large economy of "hardware" without loss of effect.

Another very useful application—one entirely different from the above—is to logic itself, but we cannot go into it here.[5] I shall merely say that,

by means of this algebra, Aristotle's treatment of categorical syllogisms has been reduced to a mere three lines, and the additional hypothetical and disjunctive syllogisms have been reduced to two more lines! Thus, all of "traditional logic," developed over the centuries and occupying tomes of literature, has been condensed to a mere *five* lines by means of this wonderful modern tool of Boolean algebra.

I hope this brief account of modern mathematics has made clear, to some extent, how very important have been the changes in mathematics since 1800, and how necessary it is for many people to become acquainted with these new methods by helping to introduce various courses, such as non-Euclidean geometry, Boolean algebra, and many others, into the curriculum of study in undergraduate schools, and even in high schools.

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SECTION OF ANTHROPOLOGY

THE CONCEPT OF CULTURE-BONDAGE*

By David French

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The term "culture-bound" has appeared on college campuses with increasing frequency, both in conversation and writing. It has been used in a way that covers, in part, a hypothesis about man which I have been exploring for some years and for which I could devise no satisfactory name. The term "culture-bondage" was suggested as the corresponding noun by A. H. Marckwardt to avoid the somewhat cumbersome "culture-boundness" he had heard me use.[1]

Among those who have used "culture-bound" in writing are: O. H. Mowrer,[2] David Easton,[3] Richard Schifter,[4] John Whiting and Irvin Child,[5] Stuart Chase,[6] Brewster Smith,[7] G. P. Murdock,[8] and Clyde Kluckhohn.[9] Kluckhohn[10] also used "nonculture-bound." V. J. Willi[11] has used "culture-boundness." It has not been possible to determine how this terminology originated. Obviously it has become quite thoroughly established.

While the terms cited above were not defined, it is possible to make inferences about their meanings from the usage. In almost every case, some criticism is intended; the authors are referring to a failure on the part of another person or group to transcend the limitations of culture. Whiting and Child[5] are an exception in using "culture-bound" to refer to ideas or terminology limited in applicability to a single society. One could call these ideas "culture-limited" if a distinction were needed. Easton's usage,[3] while much the same, occurs in a context which involves criticism of culture-bound theories.

The tentative definition to be offered here may imply the type of limitation stressed by the others, but it does not need to be used in criticism: culture-bondage is the assumption that the forms, including standards, of behavior of an individual's own society are, or should be, universal. Society, in this context, refers to that variant or section of society whose culture has been learned by the individual in question.

Most instances of the use of "culture-bound" and related terms refer to properties of ideas—the verbalized ideas of scholars. In the definition of culture-bondage above the emphasis is on assumptions, meaning that which is taken for granted, in the ideas of the scholars, for example. Many assumptions are historically derived from one's culture; at the same time, they are based on, or exist as, patterns of attitudes located

*This paper was presented at a meeting of the Section on January 24, 1955.

within individuals.

A word of explanation should be interjected here. Supporting evidence for the statements that follow has been accumulating. For example, a test for culture-bondage was devised and given to nearly the entire student body of Reed College, but the analysis of the results is not complete. The rest of the evidence is more suggestive than conclusive and will also be omitted, in large part. Space limitations are a factor here. Consequently, what follows is to be regarded as a series of hypotheses which remain to be verified, perhaps with the help of social psychologists. To two of the latter, Doctor M. Brewster Smith and Professor Herbert Hyman (as well as to Professor Howard Jolly and Kathrine French) I am already indebted for valuable advice and assistance.

The question of the origin of culture-bound attitudes can best be introduced by certain propositions about child development: children who survive are those who are aided in their maintenance and in their learning by others, usually parents, who care about them. In a different way, these children come to care or be concerned about the others. While the children learn many trivial things, they also learn, largely under the guidance of adults, to value or care about certain forms of behavior and to select them in preference to alternatives. They also become concerned with avoiding some types of behavior. They can be said to develop a series of habits which are internalized directives for behavior.

Parents are often shocked to discover great rigidity in the value judgments of children, say between the ages of 5 and 10. These children have learned a series of positive formulations about what is right and what is wrong. They are capable of being quite critical of the behavior of others, including that of the parents themselves. At this stage, the children know only a single set of rules; consequently, they do not know the customary qualifications and cannot anticipate the common contradictions which occur during the course of daily activity (*cf.* Piaget[12]).

Viewed in the light of the concept of culture, these children are incompletely enculturated. They will subsequently learn the way the members of their society customarily qualify their judgments in particular circumstances. At some point, not too many years beyond puberty, most individuals will cease to modify appreciably their attitudes toward behavior and standards, and they will thereafter accept their society's system with its modifications as "given," in an absolute sense. They are, in short, culture-bound, and it should be clear that this is a normal phenomenon.

We can assume that most people in most societies are not only initially culture-bound but will continue to be so throughout their lives. It is possible that no society could function in a stable fashion if this were not true. It seems clear, however, that the degree of culture-bondage is

a variable matter among adults in our own and in other societies. It would follow that some people have become less culture-bound in the later stages of "growing up." Such people can be said to have carried further than most the process of qualifying and understanding the assertions of their culture. In terms of the definition given above, these individuals no longer assume the universality of their culture as they know it. Nevertheless, we are probably dealing here with a question of degree, that is, with a continuum. It seems improbable that anyone could completely lack culture-bondage. When reference is made below to those who lack it, a relatively small degree of culture-bondage is meant.

It is not easy to make clear the characteristics which mark those individuals who are low in culture-bondage. I have discovered that many persons initially confuse these characteristics with others with which they are more familiar. Interestingly enough, it appears that individuals who are markedly culture-bound themselves have the greatest difficulty and, often, seem incapable of understanding the nature of the dimension under discussion.

Largely as a means of setting limits to the nature of the concept, the culture-bondage dimension will now be contrasted with other phenomena that resemble it in various ways:

(1) Those who engage in unusual and unsanctioned behavior, such as bohemians, criminals, hoboes, radicals, and various categories of marginal individuals, are not necessarily lacking in culture-bondage. The important point here is that forms of outward behavior are not, in an obvious sense, relevant to presence or absence of culture-bondage. A person who is not culture-bound may or may not conform to prevailing standards. Our concern is, rather, with his attitude toward those standards. Furthermore, most revolts and deviations concern only certain aspects of culture and are strongly patterned by the values of a society.

(2) Cynicism, apathy, indifference, tolerance, and a sense of humor about one's culture may or may not mark individuals low in culture-bondage. One might suspect that a man who writes sharp satire about the prevailing customs of his time should be classed as not culture-bound. The chances are that this is not the case, however, since most satirists accept the values or ultimate ends of their culture as universal and are primarily concerned with questioning certain current means for attaining those ends. On the other hand, a satirist may actually not be culture-bound.

(3) The possibility could be advanced that individuals who are not culture-bound are simply possessors of a particular subculture—perhaps a liberal, intellectual one—which they have learned directly from others in the same way that members of other subsocieties learn their special characteristics. A test to distinguish a person who is not culture-bound

from one who has simply learned to "think" in a similar way is this: press each for a justification for his behavior and attitudes. The one who is not culture-bound will fail to defend the ultimate "rightness" or "goodness" of his way of life.

(4) One can anticipate the assertion that culture-bondage is nothing more than a new name for ethnocentrism. These are undoubtedly related concepts. They can be distinguished, however, in this manner: ethnocentrism is a more superficial or specific manifestation of the same phenomenon, and invariably it involves a comparison between one's own social grouping and another.

(5) Lack of culture-bondage can also be differentiated from cultural relativism, despite the fact that many individuals will display both. Cultural relativism is an intellectual position on the level of ideology, while culture-bondage and its absence are attitudes or patterns of attitudes.

(6) Psychopathic personalities and undersocialized individuals are both "free," in a special sense, from the standards of behavior of their own society. They contrast with those who are not culture-bound, however, in that the latter did successfully internalize the values of their culture and did learn to take the roles of others in social situations. People who are not culture-bound are abnormal only in that they have atypical insights or understandings.

A problem of some importance, and one that is not solved easily, is the reason for differences in degree of culture-bondage among individuals. First, let us turn to the childhood environment, and, especially, to differences between families. There is probably considerable truth in the idea that most neurotics had neurotic parents; degrees of culture-bondage could be similarly perpetuated. It is possible to proceed further, however, by examining specifically the way in which questions of right and wrong are handled by parents. The behavior of the parents on both verbal and less obvious levels would be important here. When parents act toward each issue as if there were only one possible correct answer, regardless of circumstances, then the child will tend to become a culture-bound adult. On the other hand, if parents, in keeping with trends in our increasingly secular civilization, consistently give reasons for ethical decisions and stress the context of acts, as well as the morality of the acts, then the child will be potentially free of culture-bondage.

I am inclined to feel that the way in which parents approach questions of right and wrong provides a necessary but not a sufficient explanation for the degree of culture-bondage of their children. For example, playmates, teachers, and other associates can then weaken or strengthen the tendencies initiated by the parents.

It seems probable that certain kinds of cultural conditions beyond the

behavior of parents and close associates operate to reduce culture-bondage. It would be interesting to investigate the idea that there are relatively fewer culture-bound individuals in the modern Western world than in other times and places. There may also have been fewer in Greco-Roman society, as well as among certain nonliterate groups. It may be that a form of culture-consciousness can appear when knowledge of more than one cultural tradition is available. Cosmopolitan conditions, rapid social and geographic mobility, certain types of marginality, and dramatic changes in the lives of individuals could all contribute to loss of culture-bondage. In short, when there are conditions leading to a consciousness of the conventional nature of culture, the maximum number of individuals with the kind of early enculturation outlined above will lose culture-bondage.

The simple fact of the presence of more than one culture or subculture will not assure loss of culture-bondage, as is attested by the culture-bound children of many immigrant families in America. It is obvious that even individuals with habits derived from several cultural traditions can be culture-bound. One might even need to go beyond the type of enculturation and examine the individual's self esteem to reach the explanation. Members of minority groups who devalue themselves may cling even more tightly than the majority to the standards of the culture or cultures known to them.

A complete explanation for individual differences in culture-bondage would be facilitated by an adequate understanding of the personality structures at different points along the continuum. It can be anticipated that current research[13] on the authoritarian personality[14] and similar formulations will provide models of personality types which can be adapted for use in the present context. The research of David Riesman[15] also seems relevant.

The preceding pages consist of propositions about culture-bondage of a descriptive and analytic, rather than evaluative, kind. The question has not yet been raised: is it good to become free from the bondage of culture?

In a compelling section of his paper on "The Unconscious Patterning of Behavior in Society," Edward Sapir[16] argues that it is unnecessary and even mischievous for most individuals to learn about the nature, structure, or patterning of their culture. He says, in effect, that those who are not culture-bound lose a potentiality for spontaneous and imaginative utilization of the materials of culture. Sapir would limit insight to social scientists, whose business it is to undertake such analysis.

It is difficult to answer Sapir satisfactorily except to suggest that in this case he has underestimated human imagination. Just as composers and their public find their appreciation and control over music enhanced by increased understanding, so, too, can increased insight multiply the

possibilities for living one's culture.

It might be argued that culture-bondage is normal for humans, and that those who lose it become incapable of action: how can they decide anything? Or, since they have a change in the usual view of right and wrong, will they be dishonest or immoral? The facts do not seem to support either supposition. Those who are not culture-bound seem to behave much as others do except in certain special circumstances. For example, there are strong value judgments they may not make. It must not be thought, however, that they do not make decisions or value judgments. After all, they too have had the enculturating experiences of members of their society. They have a set of habits about choice-making as well as other activities. All that is really different is a special kind of insight about their own relations to their culture and its standards.

Whether or not they have thought out the implications of their activities, anthropologists and others have long been engaged in endeavoring to reduce culture-bondage—in themselves, in their students, and in the general public. If only for effective research, it has been felt necessary for anthropologists to transcend cultural limitations. More than cultural anthropology is involved. To illustrate: in physical anthropology, there has been a liberation from simplistic views in which modern men represented the goal toward which evolution had to move. Since physical ideals or standards are an aspect of culture, it is obvious that culture-bondage had been present here. It would be unwise for anthropologists ever to become complacent about their freedom from their culture. There is a constant danger of imposing unexamined categories and values derived from the anthropologist's own culture onto the data.

In the division of labor between the various disciplines dealing with man in any way, a function currently being performed by anthropology is that of pointing out culture-bound qualities in the work done in other fields. This function seems to be appropriate, and it may be the biggest single contribution that anthropology can make to scholarship in general.

Other disciplines are already partially aware of the problem of culture-bondage, and this awareness is increasing. For example, the Neo-Freudian revolution against the limitations of psychoanalysis is largely of this nature (for an explicit statement, see Fromm[17]). The uses of the term "culture-bound" cited in the writings of Smith[7] and Easton[3] occur in discussions of this problem for psychology and political science. Space limitations prevent further discussion here of the implications of culture-bondage for science. More than the social sciences are involved, since culturally derived ideas and categories can limit our understanding of any aspect of the universe.

Some educators realize that a young person's ability to appreciate the world is not necessarily increased just by gaining further knowledge.

Unless there are changes in his attitudes and in his categories of experience, a student will view new material, whether historical or contemporary, as simple projections or further examples of what he already knows. When teachers discuss "giving perspective," "broadening horizons," or especially "reducing ethnocentrism," they are often committing themselves to a reduction of culture-bondage.

In conclusion, the hypotheses have been advanced: (1) that all human beings display some degree of culture-bondage—an identification of their culture (including ideas) with universals; (2) that culture-bondage is an inevitable result of enculturation, but that certain kinds of early enculturation, coupled with facilitating cultural conditions, cause a decrease in culture-bondage after childhood. Finally, there already exists in science and in education a commitment to the reduction of culture-bondage.

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SECTION OF OCEANOGRAPHY AND METEOROLOGY

A SURVEY OF THE HURRICANE PROBLEM*

By J. Namias, Gordon Dunn, and R. H. Simpson

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(1) Introduction

R. H. Simpson

It is ironic, but true, that sometimes a war or a natural disaster is required to spark public support for research and development which holds the key to scientific progress in some fields. In 1953, public attention was dramatically drawn to the tornado menace and the warning problems it presented. In 1954, hurricanes Carol, Edna, and Hazel brought a new consciousness of the importance of the hurricane problem to residents of the Middle Atlantic and New England states. These disasters have sparked support for accelerated programs of research which are now being planned by the United States Weather Bureau. Discussions of the tornado problem by a panel of experts during the American Meteorological Society meetings at Miami, Fla., last November, set the stage for development of a large government program of tornado research, plans for which are now being completed by the Weather Bureau. Last December, an interdepartmental conference was held at the Weather Bureau to consider the hurricane problem and the various avenues of research in which accelerated programs are needed. From this conference came specific recommendations that form the cornerstone of a large government program of hurricane investigation planned for implementation later this year. Some of these plans are outlined later in this text.

The purpose of the symposium reported here is to review some of the progress already made in hurricane research and to place in perspective certain aspects of the forecast problem on which further research is needed.

(2) Tropical Cyclones Related to the Atmosphere's General Circulation

J. Namias

While the most important predictive problems concerning tropical cyclones involve time intervals of a day-to-day nature, there are many challenging questions relating to longer period fluctuations as regards the frequency and incidence of these devastating storms.

Although the tracks of tropical storms, as traced over a period of

*This symposium was presented at a meeting of the Section on January 25, 1955.

several decades appear to display little coherence when plotted on a geographical chart,[1] there are indications that, within a given fall season or for a particular fall month, storm tracks compose a more closely knit family. In other words, the spread between tracks in a given month or season appears to be less than the spread between tracks plotted for the same month of different years. The fall season of 1954, along the eastern coast of the United States, and also along the Asiatic coast, illustrate such tropical cyclone "families."

When the generalized family track characteristic of a month or season is related to the average flow pattern at mid-tropospheric levels for the same period, it appears that the envelope of tropical cyclone paths aligns itself just in advance of the planetary mean troughs, the direction of movement being fairly parallel to lines of constant anomaly (departure from long period normal). Because of this general relationship, some measure of the vulnerability of seaboard areas to hurricanes is offered by time-averaged flow patterns for mid-tropospheric levels. Also, because of the well-known fact that the upper level components (waves) of the mean planetary circulation are interdependent,[2] it is quite possible that abnormalities of tropical cyclone tracks along the eastern coasts of Asia and North America may be related. For example, over both areas tropical cyclones, in the fall of 1954, pursued paths farther west than normal in the area north of latitude 35°

It is also likely that the characteristic or mean planetary flow patterns for a given season determine the areas favorable or unfavorable for the geneses of tropical storms. The mechanism involved here might be one in which the static stability, a factor which Palmén[3] stresses in tropical cyclone formation, would be altered in areas where polar intrusions are favored (*e.g.*, the southern extremities of the preferred sites of troughs in the westerlies). In such areas, there is also an injection of cyclonic vorticity into the tropics, thus providing a favorable "circulation climate" for the growth and organization of small cyclonic vorticities into great hurricanes. Some suggestive features for this hypothesis appear in the 1954 fall patterns.

To the extent that mean flow patterns of atmospheric circulations can be predicted, it may be possible to estimate in advance the areas of greater or lesser vulnerability to tropical storms. While present techniques have shown some modest success in predicting 5- and 30-day patterns, a good deal more research is necessary before such "vulnerability" predictions can be considered reliable.

Aside from the problem of prediction, the above points are highly pertinent to any study of "hurricane cycles." One of the most significant and well-recognized features of the climatic anomaly patterns along the eastern seaboard of the United States has been the general mildness of

the winters of recent years. The characteristic anomaly (departure from long period normal) of the mid-tropospheric (700 millibars) flow pattern during the past six winters (FIGURE 1) shows that this mildness has been associated with a greater-than-normal south-to-north transport of maritime air. The mean anomaly for the past six fall seasons (FIGURE 2) also indicates a similar anomalous flow, again associated with an aberration of the planetary flow pattern in the western Atlantic. This type of anomaly also favors a more northward steering course of hurricanes north of latitude 35° and, in addition, encourages these storms to retain devastating tropical characteristics for a longer period, since the basic current has a southerly maritime fetch.

Since 1933, when fairly dense upper air data became available, the gradient of anomalous flow conducive to blocking the normal eastward motion of hurricanes has been recurring with surprising frequency. Since then, south-to-north anomalous components along latitude 40° N. have occurred in 13 of the fall seasons, while the reverse case (north-to-south anomalous flow) was observed in only 9 seasons. In the south-to-north

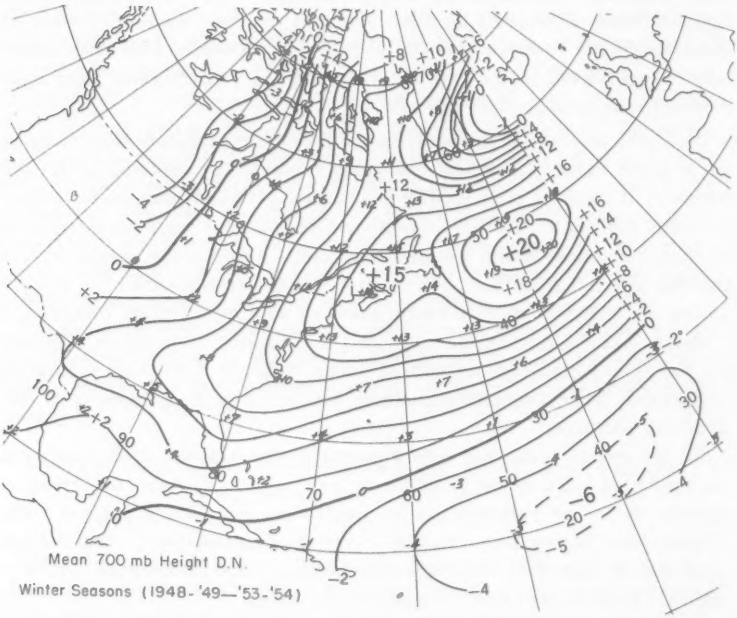

FIGURE 1. Departure from normal in the level of the 700-millibar surface (in tens of feet) for the six winters (December, January, and February) preceding 1954-55. The anomalous component of flow is directed parallel to the isolines with lower values to the left of the flow.

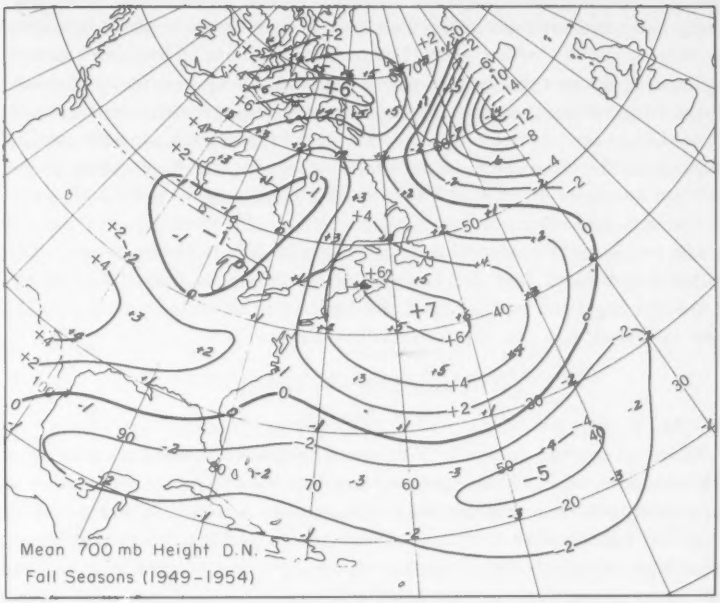

FIGURE 2. Departure from normal in the level of the 700-millibar surface (in tens of feet) for the six falls (September, October, and November) ending with 1954. The anomalous component of flow is directed parallel to the isolines with lower values to the left of the flow.

years, about twice as many hurricanes per year passed west of 65° W. (at 40° N.) than in the north-to-south years.

The ultimate causes of these anomalous mean-circulation patterns remains unknown, leaving a great void. It is not even agreed among meteorologists whether these causes are in part extraterrestrial or completely internal mechanisms of the earth-atmosphere system. Thus the prediction of climatic fluctuations in hurricanes is necessarily linked with solving the central problem, *i.e.*, the general circulation of the atmosphere.

(3) The Short-Range Forecast Problem

Gordon Dunn

The hurricane forecasting problem may be subdivided as follows: formation; detection; intensification; movement; inundations; and dissipation.

It is believed that research on hurricane formation, movement, and inundation would make the greatest and most immediate contribution to forecast improvement, although all research on tropical storms would

enhance, directly or indirectly, our ability to forecast them. Comparatively little is known about the formation of hurricanes or about how they develop so rapidly within a few hours of a coast line. The importance of forecasting accurately their direction and rate of movement is obvious. There is general agreement that any forecasting technique will require reconnaissance data from intermediate and high altitudes, both outside and inside the circulation of the storm. The accurate forecasting of the extreme acceleration of Carol 1954 must probably await the development of the new numerical prediction techniques. Hurricane tidal actions, not winds, are largely responsible for the loss of life in these storms. It is believed that tidal data and forecasting techniques are available which, if brought together, would permit the forecaster to include more detailed tidal information in his advisories to the public.

(4) Hurricane Structure

R. H. Simpson

While the general course of hurricanes seems to depend primarily upon the character of, and changes in, the planetary circulations, there is increasing evidence that the storm center does not move along a smooth path, but rather tends toward a sinusoidal or, perhaps, trochoidal movement with numerous irregularities. A forecast which does not account for these movements may err by 50 miles or more in locating the point where the storm crosses a coastline—a wide enough margin of error to mean the difference between little damage and disaster from inundations. Since these irregular movements, as well as accelerations of forward movement, depend upon the manner in which the hurricane feeds upon and utilizes the energy of its environment, their anticipation depends upon a clear understanding of hurricane structure.

Considerable progress along these lines has already been made as a result of various balloon soundings made during the approach and passage of hurricanes, and from aircraft reconnaissance of the hurricane vortex. These investigations have revealed a more complicated circulation than earlier models of the storms had envisaged, a physical explanation of which requires more quantitative information than is now available concerning distributions of momentum and of temperatures at various elevations in the storm. Some of the more important structural features that have been highlighted by data from aircraft investigations are outlined below:

(1) A small cyclonic core is found whose circulation extends upward into the stratosphere.

(2) A cluster of anticyclonic eddies is present in the upper troposphere which surrounds the cyclonic core.

(3) The core is much warmer than the surrounding storm circulation in

the lower troposphere, but is distinctly colder in the upper troposphere.

(4) In mature storms there is evidence of a dual outflow, one at the altostratus, the other at the cirrostratus level.

(5) The pressure-gradient force, while decreasing rapidly with height near the storm's eye, increases or remains constant with height in some sectors of the rain area.

(6) Spiral rainbands are associated with circulation instabilities which sometimes lead to formation of parasite eddies or false eyes.

(7) Circulations in the eye entail three levels of nondivergence: convergence occurring in the friction layer and above the rain area; a portion of the latter rising; and the remainder sinking into lower levels of the eye.

(5) Proposal For Hurricane Research

The Weather Bureau's proposal for a stepped-up program of research centers around the following projects:

(1) Use of specially instrumented aircraft: (a) to obtain detailed information on distributions of momentum, circulation characteristics, and temperature distributions at various levels, from surface to 45,000 feet in and near hurricanes in all stages of development; (b) to investigate further the structure of spiral rainbands and the nature of circulation instabilities; and (c) to test the effectiveness of using constant volume balloons in the eyes of hurricanes as a means of identifying and tracking the storm center electronically.

(2) The development of experimental models for use with electronic computers in forecasting hurricane movement.

(3) Use of coastal radar to analyze hurricane structure and hurricane micro-movements.

(4) Investigations of the nature and cause of coastal inundations resulting from hurricanes.

(5) Further studies of the role of planetary circulations in the development and recurvature of hurricanes.

(6) Further studies of variations in the incidence of hurricanes along various coastal areas and the relation of these variations to possible climatic changes.

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*DIVISION OF MYCOLOGY**

MARINE MYCOLOGY

By Helen S. Vishniac

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Ocean covers nearly three fourths of the earth's surface. Its importance has never been underestimated by man. Only comparatively recently, however, has a formal scientific interest in its microbiota led to the application to marine life of the techniques—many of them new—developed for the cultivation and study of terrestrial and fresh-water microorganisms. This interest has been motivated not only by the desire for basic knowledge of the kinds, physiology, and ecology of marine forms, but also by the desire to extend the variety and to control the amounts of food from the sea.

Marine fungi, so far as is known, are few in number and limited in variety, both compared to the other microorganisms of the ocean and to the number and variety of fresh-water and terrestrial fungi. Though few, they constitute an integral part of the marine economy. Though limited in variety, they include some uniquely interesting species. As saprophytes they not only contribute to the decay of algal remains, but are the major cause of deterioration in wood and cordage submerged in sea water under aerobic conditions (E.S. Barghoorn and D.H. Linder, 1944). As parasites of marine plants and animals, they greatly affect the availability of human food, though they are themselves inedible. The near disappearance of eel-grass along the coasts of the Atlantic, caused by a parasitic *Labyrinthula* (C.E. Renn, 1935), was a disaster to scallops and mussels and to the game birds which feed in the eel-grass community. *Lagenidium callinectes*, parasitic on the blue crab (J.N. Couch, 1942) and the "*Sirolpidium*-like" fungus which parasitizes clam and oyster larvae (H.C. Davis *et al.*, 1954) are directly attacking human food. Both parasites and saprophytes, simply because they do inhabit sea water, can be profitably used in studies of the poorly understood ecologic determinants of marine life. Parasites, in particular, because of the intimacy of the host-parasite relationship, may be expected to have developed nutritional requirements which are uniquely useful for for the study of general biochemistry. The biology of the oceans, therefore, cannot be understood or controlled without knowledge of their

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fungus flora, and such knowledge is likely to yield other dividends of basic scientific interest.

The fungus flora of the sea may be divided into two major groups, each possessing a different basic adaptation which permits marine life. The Fungi Imperfecti and Ascomycetes which have been isolated from sea water, bottom mud, or submerged wood (reviewed by C.E. Zobell, 1946) are adapted to growth in a broad range of osmotic pressures. The majority of these species are not peculiar to the sea and show no specific adaptations to an aquatic existence. While some of these isolates may be, as has often been suggested, inactive terrestrial forms deposited in the sea by wind and land drainage, Ritchie (1954) has shown that well-known terrestrial genera (including *Mucor*) are active in the sea and must therefore be considered truly marine fungi. Even those Fungi Imperfecti and Ascomycetes that are known only from marine (or estuarine) habitats and that possess spores and spore dispersal mechanisms clearly adapted to aquatic life will grow in fresh water and (though rather poorly) in triply concentrated sea-water media, as well as in normal sea water (E.S. Barghoorn and D.H. Linder, 1944). The mechanism of this wide osmotic tolerance is unknown.

The Labyrinthulas and aquatic Phycomycetes which have been investigated are, on the other hand, all stenohaline, developing only in a rather narrow range of salinities. For inquiries into the crucial points of such fit-between marine microorganisms and their environment, these obligately marine fungi are obviously more suitable than those of the first group. From what is known of the physiology of aquatic Phycomycetes, on the one hand, and of Fungi Imperfecti and Ascomycetes, on the other, the Phycomycetes seem more apt to have interesting nutritional requirements. But investigators of marine Phycomycetes, as Sparrow (1934) has pointed out, have been confronted with "a very real problem in endeavouring, first, to find sporangia which have not already discharged their zoospores in the interval between collection and examination, and, secondly, to produce conditions in the laboratory favorable for the development and discharge of these sporangia. This, together with the very small size of zoospores produced by these fungi, has greatly retarded our knowledge of the exact type of spore formed, and, consequently, our appreciation of the relationships of these organisms." It is therefore not surprising that the intermittent efforts of investigators in the past 100 years have led to the description of only about 40 species of marine Labyrinthulas and Phycomycetes, nor that nearly half of the marine Phycomycetes included by Sparrow in *"Aquatic Phycomycetes"* (1943) are considered by him to be doubtfully identified—most because of inadequate observations of the zoospores.

These same difficulties enumerated by Sparrow made the isolation in

pure culture of marine Phycomycetes by classical methods so unlikely that it did not occur. With the availability of antibiotics making possible differential suppression of bacteria, the task of isolation became relatively simple. Stanley Watson has isolated at least three species of marine *Labyrinthula* and *Thraustochytrium proliferum* by using media containing streptomycin and penicillin. I have isolated four additional holocarpic marine Phycomycetes from sea water by the same method. All of these organisms, once isolated, sporulate readily and have been easy to maintain in abundance, obviating the major difficulty of identifying collected material. Electron micrographs of zoospores, taken through the kindness of Doctor William Gaylord, have shown them to be heterokont. The paucity of other morphological characters has prevented further identification until they can be compared with fresh material isolated from known hosts infected with previously described species. Those characters most easily described from collections—extent of vegetative development, size and shape of sporangia—seem to be those most often changed on growth in pure culture. In liquid culture, isolates MF, A-11, and S-3 are all spherical bodies of approximately equal size, liberating essentially similar zoospores. They are distinguished by their morphology on agar and by their nutritional requirements, both of which are expected to be of assistance in reisolation from identified material.

Such changes in morphology are among the reasons that mycologists have correctly insisted upon the importance of observation of fungi in nature. It is also true, however, that development in any given natural host may be abnormal in the sense that that host does not present conditions for maximal development, and that conditions in pure culture can be varied at will to bring into expression whatever capacity for morphological variation a given fungus may have. I have isolated in pure culture the "*Sirolpidium*-like" fungus reported by Davis *et al.* (1954) as epizootic on mollusc larvae. Its morphology in pure culture throws considerable light upon the taxonomy of the Sirolpidiaceae. The two monospecific genera of this family, *Sirolpidium* and *Pontisma*, are separated largely by the characteristic fragmentation of the lagenidioid thalli of *Sirolpidium* to form discrete olpidioid sporangia. Additional definitive characters are the occasional branching of *Pontisma* and the occurrence of *Pontisma* on red algae, of *Sirolpidium* on green. The "*Sirolpidium*" of clam larvae (isolate Sz), however, can produce a more vigorous, more highly branched thallus than *Pontisma*, though this thallus is reduced to the single olpidioid sporangia or the unbranched tubes of *Sirolpidium* by less favorable conditions of culture. Sparrow's (1936) observations of *Sirolpidium* in infected *Bryopsis* suggested that, as Bail (1857) and Pasteur (1876) had reported for *Mucor* and other fungi, the characteristic fragmentation might be due to partial anaerobiosis. When isolate Sz is grown on agar in Petri plates, the cells do not separate, but if the plate is allowed to dry

slightly, the cytoplasm within the cells may separate into one or more olpidioid sporangia. When isolate Sz is grown in one cm. layers of liquid media, it approaches the fragmentation of *Sirolpidium* by segmenting into subthalli of two to seven cells. It is possible that a greater degree of anaerobiosis would induce even more yeastlike growth. The rather widespread occurrence of this phenomenon in fungi and the ease with which it can be manipulated makes its use as a generic character rather dubious, particularly since the natural conditions under which *Pontisma* occurs are not the same as those in which *Sirolpidium* is found The difference in host range then remains as the major definitive character of these genera. On this basis, isolate Sz is being described as a new species—of *Sirolpidium*.

The Phycomycete isolates MF, A-11, S-3, and Sz all grow well in the mineral base shown in TABLE 1, on the addition of suitable substrates, required growth factors, and (for some only) 0.1 per cent agar to provide slight physical support. This medium was developed as a nonprecipitating synthetic substitute for sea water, based upon the requirements of Watson's isolates *Labyrinthula* M and *Thraustochytrium proliferum*. It should be pointed out, however, that, of the micronutrient elements listed, only Mn, Zn, and Fe can be shown to be essential. One reason for this lies in the organic and supportive requirements of *Thraustochytrium* and *Labyrinthula* M, which, since they require amino acid substrates and 0.1 per cent agar for maximal growth under the conditions used, are not ideal subjects for studies of inorganic requirements. More definitive studies should be possible with isolate S-3, a marine Phycomycete which grows excellently when glycerol (a readily purified substrate) and thiamine are added to the mineral base.

The data obtained during the development of this mineral base nevertheless go far toward defining the determinants of the stenohaline niche in the ocean. It is suggested that this niche is determined by the requirements of marine microorganisms for NaCl, K^+, Mg^{++}, and Ca^{++} in con-

TABLE 1

MARINE MINERAL BASE

100 ml. contains:

NaCl	2.5 g.	Zn (as sulfate)	2.0 mg.
KCl	0.1 g.	Mn "	2.0 mg.
$MgSO_4.7H_2O$	0.5 g.	Fe**	0.2 mg.
KH_2PO_4	0.01 g.	Co (as sulfate)	0.02 mg.
$CaCO_3$	0.02 g.	Cu "	0.002 mg.
EDTA*	0.05 g.	B (as boric acid)	0.02 mg.
$(NH_4)_2CO_3$	0.02 g.	Mo (as sodium molybdate)	0.02 mg.

*Ethylenediamine tetracetic acid, Versene, free acid.
**As stock solution containing $FeSO_4.7H_2O$, 24.9 mg./ml. (Fe, 5.0 mg./ml.); EDTA, 30 mg./ml.; and $(NH_4)_2CO_3$, 20 mg./ml.

centrations of the order of magnitude found in sea water of chlorinity 19 o/oo. The data which support this hypothesis were obtained by varying, first independently, then in combination, NaCl, $MgSO_4 \cdot 7H_2O$, and the ionic concentration of the other elements listed. This approach seemed likely to give better results than varying total salinity, monovalent/divalent cation ratios, or Ca/Mg ratios, if, as is certainly the case for *Labyrinthula* M, there is an absolute requirement for each major element. These data may be summarized as follows (Vishniac, 1955a):

(1) NaCl: *Labyrinthulas* M, P, and A tolerate 1.5 to 3.5 or 4 per cent NaCl, with respective optima at 3, 2.5, and 2 per cent. Since the major salt of the sea is NaCl, no attempt was made to further characterize this requirement in terms of osmotic tolerances, Na^+, or Cl^- requirements. Two and five-tenths per cent NaCl gives 428 mg.-atoms/l. of Na^+ compared to 470 in sea water.

(2) K^+: The lowest concentration giving optimum growth of *Labyrinthulas* P and M was 43 mg. per cent. Isolate A is less stenohaline, being unaffected by the omission of KCl from media containing KH_2PO_4 and KOH (used in adjusting pH). The base given when brought to pH 7.8 with KOH (with gelatin hydrolysate, pH 6.0, as substrate) contains 14.4 mg. atoms/l. K^+ compared to 9.96 in sea water The K^+ concentration, over a limited range of greater than required concentrations tried, does not significantly affect the range or optimum of NaCl concentration.

(3) Mg^{++} and Ca^{++}: Maximum growth of *Labyrinthulas* M, P, and A occurred only at the highest concentrations used. These concentrations were somewhat lower than those of these ions in sea water, as permissible concentrations were limited by the requirement for keeping the medium free of precipitates. The concentrations of the mineral base give Ca^{++} 2.0 mg.-atoms/l. and Mg^{++} 20.3 mg.-atoms/l. compared with values of 10.24 and 53.57 respectively for sea water. While high absolute requirements for Ca^{++} and Mg^{++} are part of the stenohaline complex in phytoplankton as well as in fungi (L. Provasoli, J. J. A. McLaughlin, and I. J. Pintner, 1954), these ions were interchangeable to some extent for all isolates (as has also been reported for certain phytoplankton). *Labyrinthula* A, which grew over a greater range of suboptimal Ca^{++} and Mg^{++} concentrations than the more stenohaline P and M, was used in an experiment to quantitate this interchangeability. FIGURE 1 shows the equivalents of (suboptimal) Mg^{++} and Ca^{++} which allowed this isolate to grow to a density giving a Klett reading of 70 in 9 days at 20° C. Under these conditions, the equivalence of these cations depends upon which is most limiting. It should be noted that sulfate was unavoidably varied with Mg^{++}. Some of this interchangeability is undoubtedly due to the presence in the medium of metal-buffering chelating agents.

The *Sirolpidium* of clam larvae exemplifies the use of pure cultures in developing a fungus flora of the ocean. The *Labyrinthula* isolates of

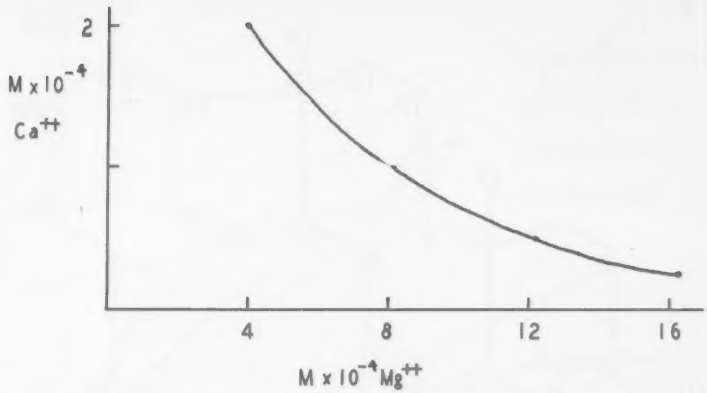

FIGURE 1. Equivalents of Mg and Ca ions per 100 ml. allowing growth of *Labyrinthula* A to Klett reading 70-71 in nine days at 20° C. (J. Gen. Microbiol. 12 No. 3).

Watson exemplify the contribution which marine fungi can make to our understanding of the inorganic determinants of marine ecology. These isolates also exemplify the unique contributions to general biochemistry which can be made by marine microorganisms. *Labyrinthula vitellina* var. *pacifica* (isolate P) requires a steroid growth factor, available in the ocean from *Zostera marina* or from its algal hosts. Steroids are best known as hormones, coordinators of metabolic activity which act on the tissue rather than the cellular level. The impression has got around that arteriosclerosis is the price we pay for (among other things) being sexual organisms—that is, that cholesterol is useful only as a precursor of bile salts and steroid hormones. The existence of five single-celled organisms with steroid growth factor requirements, which are apparently not due to the necessity for detoxifying fatty acids, implicates steroids as essential intracellular metabolites. These organisms are *Trichomonas columbae* (R. Cailleau, 1937), a flagellate; *Paramecium aurelia* (W. J. van Wagtendonk, R. L. Conner, C. A. Miller, and M. R. R. Rao, 1953), a ciliate; *Saccharomyces cerevisiae* (A. A. Andreasen and T. J. Bc Stier, 1953), when grown anaerobically on a defined medium; *Labyrinthula* P (H. S. Vishniac and S. W. Watson, 1953); and possibly pleuropneumonia-like organisms (D. G. ff. Edward and W.A. Fitzgerald, 1951). Only for *Trichomonas*, *Paramecium*, and *Labyrinthula* P is the specificity (in each case slightly different) of the requirement well worked out. The characteristics of the steroids active for *Labyrinthula* P may be summarized as follows (Vishniac, 1955b):

(1) 3β (not α)-OH or 3-keto group (see the cholesterol molecule, FIGURE 2).

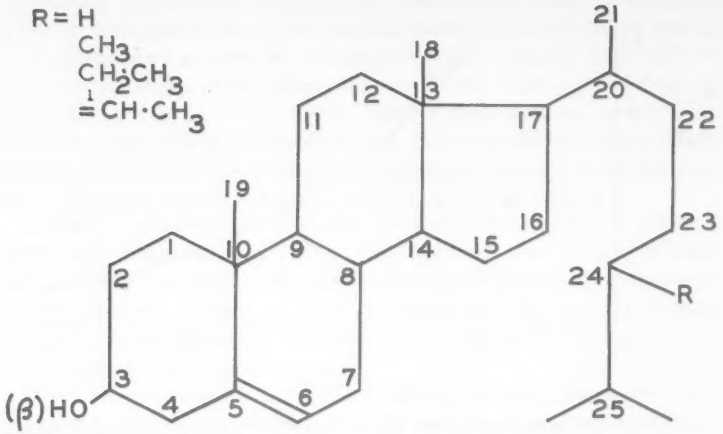

FIGURE 2. R= H, the cholesterol molecule; R= CH_3, campesterol and dihydrobrassicasterol; R= CH_2CH_3, clionasterol and β-sitosterol; R= =$CHCH_3$, fucosterol.

(2) C27, 28, or 29. C24 methyl or ethyl derivatives of active cholestenols are active whether 24a or 24b, possibly being interconverted through the (also active) vinyl derivative. Cholesterol derivatives with fewer than 27 carbon atoms are not active.

(3) Unsaturation at C4(5), C5(6), or C8(14) (not C6(7), C7(8), or C14(15)), if no other oxygen function than the 3β-OH or 3-keto group is present. $\Delta^{5,7}$(?) and $\Delta^{4,6}$ dienes are active, $\Delta^{5,22}$ dienes have slight activity.

(4) Saturated steroids with more than one oxygen function on the rings such as cholestane-3β,5α,6β-triol and cholestane-3,6-dione are active, but cholestane-3β,7β-diol and cholestane-5α-ol-3,6-dione are not.

(5) The activity of cholesterol is retained when 7-keto or 7α-OH groups are added, but not with an added 4β-OH. Δ^4-cholestenone likewise retains activity when a 6-keto or 6β-OH group is added.

The response of *Labyrinthula* P to representative steroids is shown in FIGURE 3. The differences in level of activity are probably correlated with ease of solution in the medium or of passage into the cell, rather than with ease of conversion to the essential metabolite which this requirement represents. Such differences do not therefore identify the essential metabolite; nor does comparison of the requirements of all the steroid-requiring microorganisms. Data now available show no single steroid active for all. Furthermore, the patterns of specificity differ so as to suggest that the essential metabolite may be different in each case. While *Paramecium* and *Labyrinthula* P can both use certain oxidation

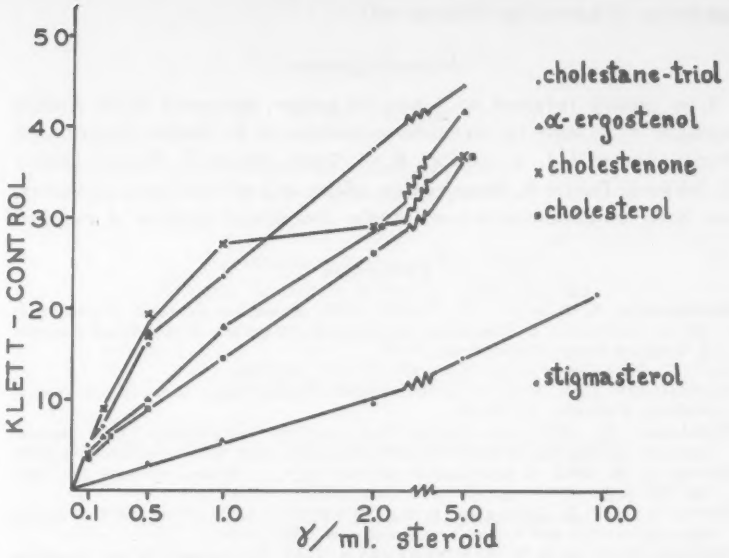

FIGURE 3. Growth response of *Labyrinthula vitellina* var. *pacifica* (P) to varying concentrations of representative steroids. Controls showed no growth (J. Gen. Microbiol. 12 No. 3).

products of active sterols and cannot use unsaturated sterols, *Paramecium* uses only C28 and C29 sterols and grows well with $\Delta^{5,22}$ dienes which are only poorly utilized by *Labyrinthula* P. *Trichomonas* and PPLO, on the other hand, do use unsaturated sterols and, so far as known, cannot utilize the oxidation products utilized by *Paramecium* and *Labyrinthula* P.

Two further lines of investigation suggest themselves as likely to identify the essential steroid metabolite which *Labyrinthula* P is unable to synthesize. First, the steroid content of organisms is a character which generally follows systematic groupings. It is therefore probable that *Labyrinthulas* M and A, which do not require steroids in the culture medium, make for themselves the same steroid which *Labyrinthula* P requires as an essential metabolite. I have therefore grown isolate M in bulk and isolated from it a sterol tentatively identified as cholesterol. There is no evidence for the presence of any other steroid. If cholesterol is the characteristic sterol of the *Labyrinthulas*, it should be present in isolate A also. Second, if cholesterol is the essential metabolite of *Labyrinthula* P, it should be possible to show that C^{14} labeled cholestenone is converted within the cell to labeled cholesterol. Such data would certainly suggest that cholesterol is, in its own right, an essential

metabolite of mammalian cells as well.

Acknowledgments

I am greatly indebted to a host of people who made these studies possible: S. E. Mills for technical assistance, S. W. Watson, Doctor S. H. Hutner, Doctor V. L. Loosanoff, H. C. Davis, Doctor K. Savard, Doctor E. Schwenk, Doctor W. Bergmann for advice and stimulating discussions, and those too numerous to mention who contributed samples of steroids.

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CONFERENCE HELD

SECTION OF BIOLOGY

BIOLOGY OF POLIOMYELITIS

Thursday, January 20 and
Friday, January 21, 1955

Conference Chairman: Karl Habel
National Institutes of Health, Bethesda, Md.

THURSDAY, JANUARY 20, 1955

POLIOMYELITIS VIRUS AND METHODS FOR ITS STUDY

Session Chairman: Karl Habel
National Institutes of Health, Bethesda, Md.

9:00 A.M. –
Introductory Remarks – Hilary Koprowski, Lederle Laboratories Division, American Cyanamid Company, Pearl River, N.Y.; Chairman, Section of Biology, The New York Academy of Sciences, New York, N.Y.

"Properties of the Virus" – Carleton E. Schwerdt, University of California, Berkeley, Calif.

Discussant: A. R. Taylor, Parke-Davis and Co., Detroit, Mich.

"Tissue Culture Technics and Their Application to Original Isolation, Growth and Assay of Virus" – Joseph Melnick, Yale University, New Haven, Conn.

Discussant: Thomas H. Weller, Children's Medical Center, Boston, Mass.

"Poliomyelitis Antibodies and Their Demonstration" –
(a) "Neutralizing Antibodies" – J. S. Youngner, University of Pittsburgh, Pittsburgh, Pa.
(b) "Complement Fixing Antibodies" – Francis L. Black, Department National Health and Welfare, Ottawa, Ontario, Canada.

Discussant: Manfred Mayer, Johns Hopkins University, Baltimore, Md.

"Tissue Culture Plaque Technic with Poliomyelitis Virus"–R. Dulbecco. California Institute of Technology, Pasadena, Calif.

SUSCEPTIBILITY OF CELLS AND ORGANISMS TO POLIOMYELITIS

Session Chairman: John F. Enders
Children's Medical Center, Boston, Mass.

2:00 P.M. –

"Comparative Susceptibility of Cells of the Same Type" – William F. Scherer, University of Minnesota, Minneapolis, Minn.

"Comparative Susceptibility of Cells of Different Types" – Sidney Kibrick, Children's Medical Center, Boston, Mass.

"Comparison of Susceptible and Resistant Cells" – Albert S. Kaplan, Yale University, New Haven, Conn.

"Relative Susceptibility of Cells in Tissue Culture and of Experimental Animals" – Herbert Wenner, Kansas Medical Center, Kansas City, Kans.

"Comparative Susceptibility of Tissue Culture Cells, Experimental Animals and Man" – George A. Jervis, Research Department, Letchworth Village, N.Y.

"Summary and Review of Cell Susceptibility" – S. E. Luria, University of Illinois, Urbana, Ill.

"Factors Influencing Susceptibility of the Host" –

(a) "Tonsillectomy and Pregnancy as They Affect Risk of Attack" Ralph S. Paffenbarger, Jr., National Institutes of Health, Bethesda, Md.

(b) Experimental Observations –

1. "Endocrinological Aspects of Pathogenesis of Experimental Poliomyelitis" – Gregory Shwartzman, Stanley M. Aronson, Constantin V. Teodoru, Max Adler, and Rene Jahiel, Mount Sinai Hospital, New York, N.Y.
2. "Viremia, Virulence and the Influence of Injections" – David Bodian, Johns Hopkins University, Baltimore, Md.
3. "Physiological Mechanisms Involved in the Localization of Paralysis" – J. Trueta, Nuffield Orthopaedic Center, Oxford, England.

POLIOMYELITIS VIRUS VARIATION

Session Chairman: Jerome Syverton
University of Minnesota, Minneapolis, Minn.

8:00 P.M. –

"Evolution of the Cellular Lesions Induced by the Poliomyelitis Virus *in vitro* as Studied with the Phase Contrast Microcinematography" – Film prepared by: G. Barski, Pasteur Institute, Paris, France.

Discussant: Hilary Koprowski, Lederle Laboratories Division, American Cyanamid Company, Pearl River, N.Y.

"Experimentally Produced Variants Using *in vivo* Technics"
(a) "In Rodents" – C. P. Li and Morris Schaeffer, Communicable Disease Center, Montgomery, Ala.
(b) "In Chick Embryo" – Manuel Roca-Garcia, Lederle Laboratories Division, American Cyanamid Company, Pearl River, N.Y.

Discussant: Hilary Koprowski, Lederle Laboratories Division, American Cyanamid Company, Pearl River, N.Y.

"Characteristics and Genetic Potentialities of Experimentally Produced and Naturally Occurring Variants of Poliomyelitis Virus" – Albert B. Sabin, Children's Hospital Research Foundation, Cincinnati, Ohio.

Discussant: John F. Enders, Children's Medical Center, Boston, Mass.

"Summary and Review of Virus Variation" – Hattie E. Alexander, Presbyterian Hospital, New York, N.Y.

FRIDAY, JANUARY 21, 1955

POLIOMYELITIS VIRUS AND THE COMMUNITY

Session Chairman: Joseph A. Bell
National Institutes of Health, Bethesda, Md.

9:00 A.M. –
"The Changing Age Distribution of Paralytic Poliomyelitis" – Carl C. Dauer, National Office of Vital Statistics, U. S. Public Health Service, Washington, D. C.

"Severity and Type of the Disease in Different Age Groups" – Dorothy M. Horstmann, Yale University, New Haven, Conn.

"Community Serum Antibody Studies" – John P. Fox, Tulane University, New Orleans, La.

Discussant: John R. Paul, Yale University, New Haven, Conn.

"A Longitudinal Study of Poliomyelitis Infection in American Families During an Interepidemic Period on a Philippine Military Base" – W. Mcd. Hammon, University of Pittsburgh, Pittsburgh, Pa.

"Virus Excretion and Antibody Response in Subclinical Cases of Poliomyelitis" – Gordon C. Brown, University of Michigan, Ann Arbor, Mich.

"Distribution of Virus Types in Clinical Cases of Poliomyelitis in the United States in 1952" – Alexis I. Shelokov, National Institutes of Health, Bethesda, Md.

Discussant: Joseph Melnick, Yale University, New Haven, Conn.

"Summary and Review of Relationships between the Virus and the Community" – Alexander D. Langmuir, Communicable Disease Center, Atlanta, Ga.

IMMUNIZATION AGAINST POLIOMYELITIS

Session Chairman: John R. Paul
Yale University, New Haven, Conn.

2:00 P.M. –
"Poliomyelitic Infection in Immunized Chimpanzees" – Howard A. Howe, Johns Hopkins University, Baltimore, Md.

Discussant: David Bodian, Johns Hopkins University, Baltimore, Md.

"Immunization of Human Beings" –

(a) "Studies on Active Immunization with Noninfectious Vaccines" – Jonas E. Salk, University of Pittsburgh, Pittsburgh, Pa.

Discussant: Albert Milzer, Michael Reese Hospital, Chicago, Ill.

(b) "Attenuated Virus Preparations" – Hilary Koprowski, Lederle Laboratories Division, American Cyanamid Company, Pearl River, N.Y.

(c) "Immunization of Chimpanzees and Human Beings with Avirulent Strains of Poliomyelitis Virus" – Albert B. Sabin, Children's Hospital Research Foundation, Cincinnati, Ohio.

"Summary and Review of Poliomyelitis Immunization" – Thomas Francis, Jr., University of Michigan, Ann Arbor, Michigan.

"Summary of Conference and Forecast: Poliomyelitis in 1984" – G. S. Wilson, Medical Research Council, London, England.

Organizing Committee:
Karl Habel, Chairman
John Enders
Howard Howes
M. J. Kopac
Hilary Koprowski
Joseph Melnick

HYDROCORTISONE, ITS NEWER ANALOGS AND ALDOSTERONE AS THERAPEUTIC AGENTS

Wednesday, January 26 and
Thursday, January 27, 1955

Conference Chairman: Joseph W. Jailer
College of Physicians and Surgeons, Columbia University, New York, N.Y.

WEDNESDAY, JANUARY 26, 1954

PHARMACOLOGY OF HYDROCORTISONE

Session Chairman: Jerome W. Conn
University of Michigan Medical School, Ann Arbor, Mich.

9:00 A.M.—

Introductory Remarks – Hilary Koprowski, Chairman, Section of Biology, The New York Academy of Sciences, New York, N.Y.; Lederle Laboratories Division, American Cyanamid Company, Pearl River, N.Y.

"Biosynthesis of Adrenal Steroids" – Gregory Pincus, Worcester Foundation, Shrewsbury, Mass.

"A Review of the Metabolism of Adrenal Cortical Steroids" – Ralph Dorfman, Worcester Foundation, Shrewsbury, Mass.

"Metabolism of Cortisone and Hydrocortisone in Man" – Ralph Peterson, National Institutes of Health, Bethesda, Md.

"The Effect of Adrenal Steroids on Renal Mechanisms of Electrolyte Excretion" – Kathleen Roberts, Sloan-Kettering Institute for Cancer Research, New York, N.Y.

"Comparison of the Metabolic Effects of Cortisone and Hydrocortisone in Man" – John C. Laidlaw, University of Toronto, Toronto, Ontario, Canada.

"The Adrenal Steroids and Secretion of Digestive Enzymes" – Burton Baker, University of Michigan Medical School, Ann Arbor, Mich.

"Use of Steroids as Anti-Inflammatory Agents" – Thomas F. Dougherty, University of Utah School of Medicine, Salt Lake City, Utah.

USE OF CORTISONE, HYDROCORTISONE AND CERTAIN SYNTHETIC STEROIDS IN SYSTEMIC DISEASE

Session Chairman: Charles Ragan
College of Physicians and Surgeons, Columbia University, New York, N.Y.

2:00 P.M.–

"Present Status of Hydrocortisone as a Therapeutic Agent in Rheumatoid Arthritis" - Edward W. Boland, Los Angeles, Calif.

"Metacortandralone and Metacortandracin in Rheumatoid Arthritis" – Joseph J. Bunim, National Institutes of Health, Bethesda, Md.

"Rheumatic Fever" – Vincent C. Kelley, University of Utah College of Medicine, Salt Lake City, Utah.

"Asthma" – Walter S. Burrage, Massachusetts General Hospital, Boston, Mass.

"Ulcerative Colitis" – Joseph B. Kirsner, University of Chicago School of Medicine, Chicago, Ill.

"Cancer" – Olof H. Pearson, Sloan-Kettering Institute for Cancer Research, New York, N.Y.

"Infectious Disease" – Laurance W. Kinsell, Institute for Metabolic Research, Highland County Hospital, Oakland, Calif.

"Allergic Reactions to Drugs" – Lawrence E. Shulman, Johns Hopkins University, Baltimore, Md.

"Connective Tissue Disease (Lupus Erythematosus Disseminatus, Periarteritis Nodosa, Scleroderma and Dermatomyositis)" – Louis J. Soffer, Mount Sinai Hospital, New York, N.Y.

"Use of Corticotropin, Cortisone and Hydrocortisone in the Nephrosis of Childhood" – Conrad M. Riley, College of Physicians and Surgeons, Columbia University, New York, N.Y.

THURSDAY, JANUARY 27, 1955

SELECTED PARENTERAL FORMS OF HYDROCORTISONE IN THERAPY

Session Chairman: A. I. Knowlton
College of Physicians and Surgeons, Columbia University, New York, N.Y.

9:00 A.M.–

INTRAVENOUS USE

"The Metabolic Effects of Intravenous Hydrocortisone and Derivatives" - Alan Goldfien, Peter Bent Brigham Hospital, Boston, Mass.

"Adrenal Virilism" – Joseph W. Jailer, College of Physicians and Surgeons, Columbia University, New York, N.Y.

"Postoperative Use of Intravenous Hydrocortisone" – Maurice Galante and Peter Forsham, University of California Medical Center, San Francisco, Calif.

"The Use of Intravenous Hydrocortisone in Asthma" – Walter S. Burrage and John W. Irwin, Massachusetts General Hospital, Boston, Mass.

"Studies of the Effect of Intravenous Hydrocortisone in Acute Hypoglycemia" – Thomas F. Frawley, The Albany Hospital, Albany, N.Y.

"Paradoxical Effects of Intravenous Hydrocortisone upon Carbohydrate Metabolism" – Herbert S. Kupperman, New York University College of Medicine, New York, N.Y.

JOINT INJECTION

"The Fate of Hydrocortisone Injected Intra-articularly" – Currier McEwen, Hildegarde Wilson, Morris Ziff, and Richard Fairbanks, New York University College of Medicine, New York, N.Y.

"The Use of Intra-articular Hydrocortisone in Arthritis" – Joseph L. Hollander, University of Pennsylvania School of Medicine, Philadelphia, Pa.

THE TOPICAL USE OF HYDROCORTISONE

Session Chairman: Marion B. Sulzberger
New York University College of Medicine, New York, N.Y.

2:00 P.M.–

SKIN

"Studies on the Absorption of Hydrocortisone Through the Skin When Applied Topically" – C. Conrad Smith, Augusta, Georgia.

"Histological Effects of Hydrocortisone on the Skin of Man" – Leon Goldman, University of Cincinnati College of Medicine, Cincinnati, Ohio.

"Topical Hydrocortisone in the Treatment of Skin Disease" – Victor H. Witten, New York University, Post-Graduate Medical School, New York University-Bellevue Medical Center, New York, N.Y.

"Topical Hydrocortisone in the External Ear" – Ben H. Senturia, Washington University School of Medicine, St. Louis, Mo.

EYE

"The Use of Topical Hydrocortisone in Corneal Injury and Diseases of the Uveal Tract" – Dan M. Gordon, Cornell University Medical College, New York, N.Y.

"The Use of Topical Hydrocortisone in Inflammatory Diseases of the Conjunctiva, Sclera and Cornea" – Ellis H. Steffensen, Henry Ford Hospital, Detroit, Mich.

NOSE

"Controlled Studies in the Topical Application of Hydrocortisone in Vasomotor Rhinitis" – John H. Burger, Henry Ford Hospital, Detroit, Mich.

HALOGENATED ANALOGS OF HYDROCORTISONE

Session Chairman: George W. Thorn
Peter Bent Brigham Hospital, Boston, Mass.

8:00 P.M.–

FLUOROHYDROCORTISONE

"Biological Effects of Flurohydrocortisone and Related Halogenated Steroids in Animals"–Josef Fried, The Squibb Institute, New Brunswick, N. J.

"Biological Effects of Fluoronated Derivatives of Hydrocortisone and Progesterone in Man" – Albert Renold, Peter Bent Brigham Hospital, Boston, Mass.

"Experiences with 9α Fluorohydrocortisone Acetate in Patients with Rheumatoid Arthritis" – Edward W. Boland, Los Angeles, Calif.

"Comparative Effectiveness of Fluorohydrocortisone and Hydrocortisone in the Treatment of Skin Disease" – Marion B. Sulzberger, New York University School of Medicine, New York, N.Y.

ALDOSTERONE

"The Possible Role of Aldosterone in Edematous States" – Ben Johnson and John Luetscher, Stanford University, Palo Alto, Calif.

"Comparative Effects of Aldosterone and Fluorohydrocortisone" – George Thorn, Peter Bent Brigham Hospital, Boston, Mass.

"The Clinical and Physiological Effects of Aldosterone and 9α Fluorohydrocortisone and Some Newer Steroids" – E. Ward and P. Hench, The Mayo Clinic, Rochester, Minn.

NEW MEMBERS

Elected January 27, 1955

LIFE MEMBERSHIP

Nachtigall, Henry B., M.D., Internal Medicine. Private Practice, New York, N.Y.

SUSTAINING MEMBERSHIP

Beck, David, M.D., Internal Medicine. Consulting Physician, Mt. Sinai Hospital, New York, N.Y.

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Friedlander, Jack, D.D.S., Biology. Private Practice, New York, N.Y.

Greer, Alvis E., M.D., Pulmonary Mycology. Owner, Director, The Greer Clinic, Houston, Texas.

Gregory, J. E., M.D., Mycology. Private Practice, Pasadena, Calif.

Gutmann, Max, M.D., Biology. Private Practice, Brooklyn, N.Y.

Helfer, Lawrence S., M.D., Psychiatry. Private Practice, New York, N.Y.

Hoffmann, Richard H., M.D., Psychiatry. Consulting Neuro-Psychiatrist, New York, N.Y.

Jue, William Poy, M.D., Internal Medicine. Private Practice, Oakland, Calif.

LeMunyan, Cobert Duane, B.S., Animal Behavior. Assistant Head, Research Animal Laboratories, Naval Medical Research Institute, Bethesda, Md.

Reader, Samuel, Tissue Culture. President, Microbiological Associates, Inc., Bethesda, Md.

Simon, David L., M.D., Medicine. Research Fellow in Cardiology, University of Cincinnati, Cincinnati, Ohio.

Vincent, Monroe, Tissue Culture. Microbiological Associates, Inc., Bethesda, Md.

Whipple, Robert L., Jr., M.D., Human Emotions. Assistant Clinical Professor, Medicine, Emory University, Ga.

Woodin, William G., M.D., Allergy. Associate Clinical Professor, Medicine, State University of New York, Syracuse, N.Y.

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Amster, Milton W., M.D., Medicine. Director, Medical Service, Pfizer Laboratories, Chas. Pfizer and Company, Brooklyn, N.Y.

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Ard, Robert W., M.D., Pharmaceuticals. Medical Department, Pfizer Laboratories, Chas. Pfizer and Company, Brooklyn, N.Y.

Arluck, Edward Wiltcher, Ph.D., Clinical Psychology. Assistant Professor, Psychology, City College of New York, New York, N.Y.

Aronson, Joab K., Ph.D., Muscle Physiology. Instructor, Department, Physiology, Chicago College of Osteopathy, Chicago, Ill.

Aserinsky, Eugene, Ph.D., Neurophysiology. Instructor, Physiology Department, Jefferson Medical College, Philadelphia, Pa.

Aspiotis, Ar. A., M.D., Neurology, Psychiatry. Director, Institute of Medical Psychology, Boston City Hospital, Boston, Mass.

Bard, Morton, Ph.D., Psychology. Research Fellow, Sloan Kettering Institute for Cancer Research, New York, N.Y.

Behrend, Hans J., M.D., Physical Medicine. Attending Physical Medicine, Hospital for Joint Diseases, New York, N.Y.

Bennett, Ivan Frank, M.D., Psychiatry. Assistant Chief, Acute (Intensive Treatment) Service, Veterans Administration Hospital, Coatesville, Pa.

Benzinger, Theodor H., D.Sc., Physiology. Head, Bio-Energetics Division, Naval Medical Research Institute, Bethesda, Md.

Berenson, Gerald S., M.D., Cardiology. Assistant Professor, Department, Medicine, Louisiana State University, New Orleans, La.

Berkow, Bori, M.D., Internal Medicine. Associate, Medicine Staff, Perth Amboy, N.J.

Bird, Joseph G., Ph.D., Clinical Pharmacology. Clinical Pharmacologist, Sterling Winthrop Research Institute, Rensselaer, N.Y.

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Borst, Lyle, Ph.D., Nuclear Physics. Chairman, Department, Physics, New York University, New York, N.Y.

Bostrom, Roland C., B.S., Medical Physics. Project Engineer, Huntington Station, N.Y.

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Denkewalter, Robert G., Ph.D., Chemistry. Director, Provess Research and Development Division, Merck and Company, Rahway, N.J.

Dettwiler, Herman Andrew, Ph.D., Viruses. Director, Biological Production Division, Eli Lilly and Company, Indianapolis, Ind.

Diacumakos, Elaine G., B.S., Cellular Physiology. Graduate Student, New York University, New York, N.Y.

Douglas, James M., M.Sc., Virus Research. Biologist, Lederle Laboratories, Pearl River, N.Y.

Dubnick, Bernard, Ph.D., Biochemist. Senior Scientist, Warner-Chilcott Research Laboratories, Morris Plains, N.J.

Dugal, Louis Paul, Ph.D., Experimental Physiology. Professor, Leval University, Quebec, P.Q., Canada.

Dumas, Kenneth J., M.D., Medicine. Medical Department, Chas. Pfizer and Company, Brooklyn, N.Y.

Du Pree, Alphonse Leure, M.S., Biochemist. Research Biochemist, Rockland State Hospital, Orangeburg, N.Y.

Elveson, Leon, B.S., Medical Literature. William Douglas McAdams, Inc., New York, N.Y.

Espenshade, Marlin A., Ph.D., Microbiology, Mycology. Research Mycologist, Wyeth Laboratories, Inc., West Chester, Pa.

Ewing, James Halsted, M.D., Neoplasia. Assistant Attending Physician, Memorial Hospital, New York, N.Y.

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